

1 SCIENTIFIC REVIEW PANEL ON TOXIC AIR CONTAMINANTS
2 STATE OF CALIFORNIA
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6 PUBLIC MEETING
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13 April 27, 2001
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ALSO PRESENT:
Dr. Mark Miller
Dr. David Morry

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1 CHAIRMAN FROINES: I would like to call to
2 order the Scientific Review Panel meeting for
3 April 27th, 2001 officially.

4 The first item to -- for discussion is not on
5 the agenda, and it's meant as a very informal comment by
6 Dr. Byus on the progress with respect to the
7 organophosphate document.

8 DR. BYUS: Thanks, John.

9 CHAIRMAN FROINES: And no action will follow
10 from this update.

11 DR. BYUS: They were proceeding -- I've
12 received two of the chapters so far and have had
13 conference calls on both of them. That's proceeding
14 quite well. They've updated their schedule to me, which
15 I gave to the panel, Jim and to John.

16 And so we're proceeding quite quickly on those
17 documents. It looks like we're going to meet the
18 schedule that they had originally given us. So that's
19 all they wanted me to tell you at the meeting since they
20 couldn't be here.

21 CHAIRMAN FROINES: The second item -- I've
22 passed out -- I think everybody -- if there's anybody
23 who's missing a copy, please let me know -- of the draft
24 agenda for the scientific meeting on issues in the
25 assessment of health impacts of gasoline emissions in

1 California, which is scheduled for June 12th and 13th
2 this year. And I think it's an absolutely outstanding
3 agenda, so we would urge interested scientists and
4 professionals to attend the meeting. It's sponsored by
5 OEHHA, and it will be held at UCLA.

6 After those bookkeeping, I am going to turn the
7 meeting at this point over to Melanie, Dr. Melanie
8 Marty, to discuss the children's environmental health
9 compounds.

10 DR. MARTY: Good morning. Is this mike on?

11 DR. GLANTZ: Yeah.

12 CHAIRMAN FROINES: I think -- let me just give
13 the ground rules. I think what we're going to do --
14 Melanie, tell me if you don't agree -- we're going to --
15 Melanie is going to present the criteria and give an
16 overview of the process to begin with, and then we can
17 have questions during that time and subsequent to it,
18 and then we'll proceed to address the individual
19 chemicals on a one-by-one basis.

20 We haven't assigned lead status to anyone on
21 the panel for a particular chemical, so as we are
22 discussing a particular chemical, we'll go around the
23 room and have input from the panel in order.

24 So is that your sense of it --

25 DR. MARTY: Yes.

1 CHAIRMAN FROINES: -- Melanie? Go ahead.

2 DR. MARTY: Okay. Today we're going to talk
3 about a document we drafted, the prioritization of toxic
4 air contaminants, under Senate Bill 25, which is the
5 Children's Environmental Health Protection Act.

6 I just thought I'd start -- next slide, please.
7 The miracles of modern technology.

8 (Pause.)

9 DR. MARTY: Okay. I thought I'd start with
10 just some quotes from the statute about what we're
11 supposed to be doing. The office, which is OEHHA, in
12 consultation with the state board, which is the Air
13 Resources Board, shall establish a list of up to five
14 toxic air contaminants -- and these were specifically
15 that had already been identified under existing
16 statutes -- that may cause infants and children to be
17 especially susceptible to illness.

18 In developing the list, the law requires us to
19 take into account public exposures to toxic air
20 contaminants, whether by themselves or interacting with
21 other toxic air contaminants or criteria air pollutants.

22 And then there were four specific factors that
23 the law requires us to evaluate. Next slide, please.
24 These factors include exposure patterns among infants
25 and children that are likely to result in

1 disproportionately high exposures; special
2 susceptibility of infants and children to air pollutants
3 in comparison to overall general population; the effects
4 infants and children of exposures to TACs and other
5 substances with a common mechanism of action; and,
6 finally, the interaction of multiple pollutants,
7 including the interactions between criteria pollutants
8 and toxic air contaminants.

9 CHAIRMAN FROINES: Just one point. Andy, could
10 you go back to the previous slide?

11 I think it's important to bring to the panel's
12 attention that the -- that the words on the -- under the
13 first bullet are "that may cause infants and children to
14 be especially susceptible to illness." So the word
15 "may" of course is a problem in some respects because it
16 is -- it doesn't define the scientific rigor associated
17 with that decision. So the panel needs to be aware of
18 that designation.

19 Sorry.

20 DR. MARTY: What happens after OEHHA
21 establishes this list is that the ARB steps in. They
22 must within two years evaluate existing control
23 measures. Those are the airborne toxic control
24 measures, or ATCMS, for substances on the list and
25 revise them, if appropriate.

1 If there is not an ATCM for a substance that
2 gets on the list, then ARB within three years must
3 prepare what they call a "needs assessment" or a report
4 on the need for regulations for those TACs and adopt
5 them if it's appropriate.

6 DR. GLANTZ: Melanie, I just have a question.
7 Of the 11 compounds that you suggested in the Tier 1 and
8 Tier 2, are there any of them that ARB doesn't have
9 toxic control measures for?

10 DR. MARTY: Yes.

11 DR. GLANTZ: Which ones?

12 DR. MARTY: I was afraid you were going to ask
13 me that.

14 DR. GLANTZ: I mean, it's not -- it's a little
15 bit off the subject, but I'd be just curious.

16 DR. MARTY: Okay. And ARB can correct me if
17 I'm wrong. Formaldehyde, lead, polycyclic aromatic
18 hydrocarbons, acrolein, glycol, ethers. They're working
19 on diesel. Mercury there is not one, PCBs or vinyl
20 chloride. So --

21 CHAIRMAN FROINES: Dioxins?

22 DR. MARTY: -- most of them. Dioxins, there is
23 a control measure from -- for emissions from medical
24 waste incinerators.

25 DR. GLANTZ: Okay.

1 CHAIRMAN FROINES: Can somebody write down that
2 list and give it to the panel?

3 DR. GLANTZ: Well, it's in the transcript.

4 CHAIRMAN FROINES: No. I mean the ones that
5 don't have control measures.

6 It's all right. Go ahead.

7 DR. MARTY: Next slide, Andy.

8 What I'd like to do now is talk about our
9 prioritization activity, how did we start with over 200
10 TACs, depending on how you count them, and work down to
11 the list of 11 and then the list of 5 proposed. We
12 started with a list of all 200 TACs, and we actually
13 have that list if the panel wants it to go through it.

14 Peter, do you want to hand out those lists?

15 We started with a list of TACs. And, actually,
16 it was a summary table from ARB's prioritization process
17 that they used to come up with candidates for us to look
18 at health effects. So we started out with the TACs and
19 information on ambient concentration data.

20 We updated that data, if there were new data
21 available from the ARB's monitoring network, for
22 example. We divided the ambient concentration data by a
23 chronic reference exposure level and then ranked the
24 chemicals in order of that ratio.

25 What this does is gives you an indication of

1 where the ambient concentrations that have been measured
2 are with respect to a benchmark that you consider a safe
3 dose. So that's what we tried to do to see, okay, are
4 any of these actually above our chronic reference
5 exposure level, or are any of them close to our chronic
6 reference exposure level?

7 We also wanted to deal with the carcinogenicity
8 piece, so we multiplied the ambient concentration data
9 by available unit risk factors to rank the carcinogens
10 by ambient cancer risk.

11 The chemicals -- since we're not charged with
12 having a list of carcinogens and a separate list for
13 non-carcinogens, we have to combine those two rankings.
14 So the chemicals were placed on single lists, and
15 depending on the ratio of the ambient data to the REL or
16 the product of the URF times the ambient concentration,
17 they were moved up or down in the ranking according to
18 which really drove the risk for that chemical.

19 DR. FRIEDMAN: Could you give a numerical
20 example of that to make it a little bit clearer?

21 DR. MARTY: You know what?

22 CHAIRMAN FROINES: I can.

23 DR. MARTY: Table --

24 CHAIRMAN FROINES: I can.

25 DR. MARTY: Okay.

1 CHAIRMAN FROINES: Acrolein has a -- in their
2 document has a 14.3 microgram per cubic meter is the
3 most recent exposure level. It is when you take that
4 value and divide the air concentration by the REL, you
5 get a ratio of 238. Whereas the ratio for formaldehyde
6 is 1.1. The ratio of arsenic is .5 and then toluene,
7 for example, is .025, so that -- methyl chloroform, for
8 example, goes to .0005, so there's a very wide range of
9 air concentrations relative to the REL value, and
10 there's a lot of air concentrations that are missing.

11 DR. FRIEDMAN: Well, the question I had was not
12 that but how you merged the cancer potency and that
13 ratio. How you -- you know, how you then ranked -- came
14 up with the ranking on a single list.

15 DR. MARTY: Okay. The -- there were only
16 really a few cases where it was obvious. If that ratio
17 of the ambient concentration to the REL was extremely
18 small, who cares?

19 DR. FRIEDMAN: Right.

20 DR. MARTY: So the cancer risk would drive it
21 in that case.

22 DR. FRIEDMAN: So you -- in other words, if the
23 cancer risk was bigger than that other ratio, you
24 selected that as the number with which to rank all the
25 chemicals?

1 DR. MARTY: Yes. In a sense. They're
2 different -- they're measures of different things, so
3 there is some judgment that you have to use: Is that
4 cancer risk more of a concern than the reference
5 exposure level? And generally the answer to that is yes
6 because the thresholds are assumed -- that are assumed
7 for non-cancer endpoints means that if you're below that
8 REL, you're pretty -- pretty confident that it's an okay
9 exposure to be -- an okay concentration to be exposed
10 to.

11 Whereas the cancers are assumed to be linear
12 related, so what you have there is you have a
13 probability of cancer risk. And it may mean that
14 neither of them is really very important, but if one was
15 more important than the other, it would push up in the
16 ranking.

17 DR. FRIEDMAN: And when you say "more
18 important," do you mean a higher -- just a higher ratio?

19 DR. MARTY: A high -- a ratio that would, for
20 example, approach .5 or even .1 for the ratio of the
21 concentration to the REL. That to me would be more
22 important than something that had a cancer risk of 10 to
23 the minus 8. So it's -- it's because --

24 DR. FRIEDMAN: Was there ever a case when the
25 cancer risk moved the chemical up higher on the ranking?

1 DR. MARTY: I'm sure that's true.

2 DR. MORRY: There are one or two cases like
3 that. So we had two lists. One ranked by cancer risk,
4 one ranked by non-cancer endpoints. A lot of chemicals
5 were the same on both lists, and, in general, the order
6 was the same where the chemicals appeared on both list.
7 It was just a matter of putting them in register with
8 each other.

9 And in a few cases you've got to decide, well,
10 you've got some non-cancer values and some cancer values
11 that are sort of in the same part of the list, and which
12 one do you put above -- which chemical do you put above
13 another chemical? So there's a little bit of
14 arbitrariness in doing that. But it's just -- the
15 arbitrariness would only affect up or down.

16 DR. MARTY: Judgment day. A little bit of the
17 judgment.

18 DR. MORRY: Okay. But the judgment would only
19 taint it up or down, like, a few positions.

20 DR. FRIEDMAN: I see. I think, you know, in
21 some of the comments, the public comments, about the
22 lack of transparency, I think this is one area where it
23 isn't totally transparent especially when you say that,
24 you know, there's a matter of judgment there, and the
25 criteria aren't quite clear.

1 DR. MARTY: I think what we want to do is -- go
2 ahead, Stan.

3 DR. GLANTZ: Well, I had a long discussion with
4 Melanie and her people about this. I agree that several
5 of the public commenters commented on the lack of
6 transparency, and I think that's a problem. I think
7 that the process isn't quite as irrational as it looked,
8 the way they described it, when I sat down and had them
9 explain it.

10 And what I would propose doing is let Melanie
11 finish talking, this part of the talk where you're just
12 talking about the prioritization, and then I think we
13 should just stop and discuss that and then go on to the
14 other chemicals.

15 CHAIRMAN FROINES: I think it's important -- I
16 think it's important to have a specific discussion --

17 DR. GLANTZ: Yeah.

18 CHAIRMAN FROINES: -- about the document and --

19 DR. GLANTZ: Yeah. I mean, I --

20 CHAIRMAN FROINES: -- and the methodology.

21 DR. GLANTZ: Yeah. I mean after discussing it
22 with them, I think the prioritization procedure that
23 they used was pretty reasonable, but the way it was
24 described, it was completely opaque. And so what I'd
25 like to do is just let her finish this part of it, and

1 then I think we should discuss this and bring out
2 exactly how it was done.

3 And I had given Melanie a couple of suggestions
4 of ways I think it ought to be presented, which would, I
5 think, make people a lot more comfortable.

6 DR. MARTY: I actually prepared some tables at
7 the request of Stan, which will probably shed some light
8 onto this and which we can put into the document when we
9 revise it and make it clearer of what it is that we
10 actually did.

11 DR. GLANTZ: Why don't you just finish this
12 part of the presentation.

13 CHAIRMAN FROINES: I have one question, if
14 Peter is in the room or Jim. Do we have access to a
15 Xerox machine because I do have the document that lists
16 all these values?

17 DR. MARTY: We have that as a handout.

18 CHAIRMAN FROINES: You do. Okay.

19 DR. MARTY: So we need to get -- and you --

20 DR. BYUS: You must have sent it to us.

21 DR. MARTY: Yes. We sent that to the panel
22 with the document.

23 DR. GLANTZ: Yeah. But I actually don't find
24 that as helpful as -- why don't -- just let her finish.

25 CHAIRMAN FROINES: Let's go ahead. But just so

1 for everybody on the panel, you have the document.

2 DR. GLANTZ: Well, that is a document, but I
3 personally didn't find that -- that's not what I think
4 should be presented. But, anyway, why don't, Melanie --
5 why don't you just finish this part of the presentation,
6 and then we can begin pondering.

7 DR. MARTY: Okay. There were some chemicals
8 that had unit risk factors but no reference exposure
9 levels, so we dealt with those by, again, multiplying
10 the unit risk factor by the ambient concentration data,
11 and then we positioned those tables according to the
12 product of that with respect to the other carcinogens in
13 the table.

14 Next slide, please. This initial procedure
15 provides a ranking based on existing health criteria,
16 existing reference exposure levels and existing unit
17 risk factors and the ambient concentration data. Since
18 there are some chemicals for which there are no ambient
19 concentration data readily available, we wanted to look
20 at other indications that there is exposure in
21 California.

22 So we evaluated other sources of exposure
23 information, which included the Air Toxics Hot Spots
24 emissions inventory database. There's over 30,000
25 facilities in that database, and the emissions are not

1 something that you can readily translate into a
2 concentration, but you can get an idea in terms of
3 pounds per year of how much of these chemicals are being
4 emitted by facilities in California.

5 We also looked at the mobile source emissions
6 database to get an idea of which chemicals from a mobile
7 source perspective are important.

8 After doing that, we still needed to consider
9 toxicological properties and whether or not there is a
10 known sensitivity of young organisms relative to old
11 organisms, old people, adults, for that particular toxic
12 chemical. So we also took that into consideration.

13 Andy, I think I'm on the next slide.

14 DR. MARTY: So we took into consideration --

15 DR. SALMON: Sorry.

16 DR. MARTY: Go back one. We took into
17 consideration the emissions inventories from mobile and
18 stationary sources. We reviewed the entire list of
19 TACs, not just those that had ambient concentration
20 data, to look for any chemicals with known toxicological
21 properties that would be of concern. For example,
22 mercury, we didn't have good ambient concentration for
23 mercury.

24 And over half of the TACs dropped out at this
25 point.

1 DR. BLANC: Because?

2 DR. MARTY: Because they either had -- Table A,
3 which is being passed out to you, is a list of all the
4 TACs.

5 DR. BLANC: Right.

6 DR. MARTY: Table B is a list of the chemicals
7 that dropped out at this stage of the game.

8 DR. BLANC: Because you did not have ambient
9 data.

10 DR. MARTY: We didn't have either ambient data
11 or any information on emissions, and/or we did not have
12 unit risk factor or chronic level exposure levels. So
13 for some of those, all of those apply.

14 DR. BLANC: Doesn't the actual legislation
15 refer to exposures or potential exposures in its
16 language?

17 DR. MARTY: Well, I have the statute in front
18 of me.

19 DR. BLANC: Potential and --

20 DR. MARTY: The potential exposures would be
21 taken care of by looking at emissions inventory data.
22 Is this stuff even emitted in California? Is it an
23 airborne chemical in California?

24 DR. BLANC: Well, let me ask you if you thought
25 something was about to enter into the marketplace on a

1 mass scale, wouldn't that represent a potential
2 exposure, or if something for which you don't have
3 quantified release data and yet you would know from some
4 other source that it must be released? Wouldn't that
5 be --

6 DR. MARTY: Well, we did talk to the Air
7 Resources Board to get at precisely those issues, but
8 there were no hard data to go on to take care of those
9 contingencies.

10 DR. BLANC: Well, wouldn't that, in fact, be an
11 area where there wouldn't be hard data, or what does
12 hard data mean to you in that situation?

13 DR. MARTY: Either an indication of -- they're
14 an emission inventory, pounds per year from a certain
15 facility, or something that's been looked at from the
16 mobile source side of things in terms of the mobile
17 source emissions inventory. Neither of those
18 inventories is perfect.

19 For example, the hot spots facility emission
20 inventory, they inventory about 425 chemicals. Some of
21 those emissions estimates are just that. They're
22 estimates. They're based on throughput of the facility.
23 They're based on use by the facility, and those are not
24 perfect estimates of emissions by stretch.

25 And there may be chemicals which are not being

1 reported. Although, in this case, for the TACs, they're
2 all substances which need to be reported under the Air
3 Toxic Hot Spots.

4 In the case of the mobile source emissions
5 inventory, yes, we know about benzene and butadiene and
6 formaldehyde and the more common chemicals that we're
7 concerned about from mobile sources, but there may be
8 some that no one's looking at.

9 DR. BLANC: Well, if you don't mind, let me
10 just ask the specific case examples that I can -- as a
11 way of clarifying your thinking. There is a lot of
12 concern about the potential introduction of organified
13 manganese as a gasoline additive, as you're aware.
14 Manganese is a neurotoxin for which there would be a lot
15 of rationale for considering --

16 DR. MARTY: Yes.

17 DR. BLANC: -- pediatric sensitivity.
18 Manganese is a -- manganese and manganese compounds are
19 TACs. By what criterion would one eliminate or not
20 eliminate manganese from being on the list of things to
21 be given a great deal of consideration?

22 DR. MARTY: We actually put manganese into the
23 top 35 that we did literature reviews for, for precisely
24 that reason.

25 DR. BLANC: And then what happened?

1 DR. GLANTZ: Well --

2 DR. MARTY: Let me get there.

3 DR. BLANC: So -- but by your criteria, that

4 wouldn't be --

5 DR. GLANTZ: Having spent a lot of time giving

6 Melanie and her staff a very hard time about this, I

7 really think we would have a more productive discussion

8 if you let her just finish describing what they did.

9 DR. BLANC: Well, that was the nature of my

10 question. I wanted to understand the process by

11 focusing on the sample, and I'm going to actually

12 keep -- I'm going to continue, over the course of the

13 morning, be returning to specific examples so that I can

14 understand how those fit into your process.

15 DR. MARTY: Sure.

16 Okay. Does the panel have the tables yet?

17 CHAIRMAN FROINES: Yeah, we do.

18 DR. MARTY: Table A is just a list of the TACs,

19 so you can put that on the bottom of your pile now.

20 Table B is a list of the chemicals that fell out because

21 there were no indications of exposure either from

22 emissions inventories or ambient concentration data, or

23 there were no health criteria, no RELs, no unit risk

24 factors.

25 DR. GLANTZ: I have one -- I'm not breaking my

1 own rule, but I just have a question on a case.

2 CHAIRMAN FROINES: Yes, you are.

3 DR. GLANTZ: No, I'm not.

4 DR. BYUS: You are.

5 DR. GLANTZ: Only Dr. Freud is allowed to do

6 that. Anyway, when you say for which there are no RELs,

7 cancer potency factors and adequate ambient air levels

8 data, does that mean that if you didn't meet -- what if

9 you had something that was like -- had huge cancer

10 potency but there was no REL? That wouldn't drop out?

11 DR. MARTY: No. That didn't. That wouldn't

12 drop out.

13 DR. GLANTZ: So that's really -- so I think --

14 so you're -- given the sensitivities about how this list

15 was made, I mean, I think we need to be very precise

16 here. So of the stuff in Table B, of however many are

17 in here, 137 compounds here, how many of these are on

18 this list because you couldn't find any evidence of

19 exposure in California?

20 DR. MARTY: I would say the vast majority.

21 DR. ATKINSON: Many of those are probably

22 either constituents of gasoline or some are formed in

23 the atmosphere where there's really going to be

24 exposure. But there may be no actual emissions data; is

25 that right?

1 DR. MARTY: That's a problem, yes. Yeah. And
2 many of these do not have health values, so there's no
3 handle on the toxicity in a quantitative sense.

4 DR. GLANTZ: Well, I think one thing, again,
5 and getting to the point of making this as transparent
6 as possible, I would suggest that you break Table B up
7 into pieces, and I would have -- the ones for which you
8 have no evidence of emission, that is one list. So you
9 can say to people, we excluded these -- not withstanding
10 what Roger just said, because we couldn't find any
11 evidence that it's being released into the air and then
12 that's very clear; okay? That that's why you're not
13 looking at those.

14 And then I think if -- for the ones where you
15 have no data documenting health impacts, I would have
16 that as a separate sublist.

17 DR. MARTY: Okay. I wouldn't say that there
18 were no data --

19 DR. GLANTZ: Well --

20 DR. MARTY: -- documenting health impacts, but
21 rather there was no quantitative assessment of those
22 chemicals.

23 DR. GLANTZ: Okay.

24 DR. MARTY: There are chemicals that initially
25 were on Table B that we moved up because of concerns

1 about --

2 DR. GLANTZ: Okay. But that's moving it -- I
3 mean, the question -- the concern I think is what are
4 you dropping off the list and why? And then we'll
5 get -- when you get down to the short list, that's, of
6 course, where the biggest debates come. But I think to
7 just say we didn't include these because there was no
8 evidence of emission, and then the rest of these, within
9 this list, which ones you didn't include because you
10 didn't have a unit risk or a REL. And that way it's
11 very clear why these are not here.

12 Now, that doesn't mean that, if you look at the
13 point that Paul made, that if something's about to be
14 emitted, you could put it on a higher list. But, you
15 know, that at least explains where this list came from.

16 I really think -- I mean, when I read through
17 all the comments, this issue of making the process
18 really transparent is absolutely crucial for people, you
19 know, buying into this document. And the -- and it's --
20 I realize when you go from 200 to 5 or 11 and given that
21 there's apples and oranges aspects of this, you do have
22 to apply some judgment. But I just think the more
23 explicit you can make all of that, the more comfortable
24 people will be with the outcome. So that would be my
25 suggestion for this.

1 DR. MARTY: Okay.

2 DR. GLANTZ: So now I'll let you go ahead with
3 the presentation.

4 CHAIRMAN FROINES: I think, as a generality, a
5 point that needs to be made is that any member of the
6 public should be able to look at the 200 TACs and
7 understand why it's where it is on the list because I
8 think -- I think these lists are not adequate at this
9 point and -- but at some point in the future we just
10 need to make sure that anybody in the audience can pick
11 it up and say, "Oh, I may not agree with why this is
12 where it is, but I understand why" --

13 DR. MARTY: Why it's where it is.

14 CHAIRMAN FROINES: -- "it's where it is."

15 DR. BLANC: So, just to clarify again for the
16 specific chemicals, I understand as it relates to your
17 comment that I can't tell from this list why something
18 fell out, but parathion, for example, is that because
19 it's no longer in use? It's a banned pesticide.

20 DR. MARTY: Actually, there were two issues
21 there. One is with pesticides in general. This statute
22 only applied to the TACs that were not pesticides. In
23 other words, they did not --

24 Jim, can you help me out here?

25 DR. BLANC: It doesn't say that in the

1 legislation explicitly. It says something obliquely.
2 Have you had legal counsel actually make that extremely
3 clear?

4 DR. MARTY: ARB's legal counsel made that --

5 DR. BLANC: In writing and that's included in
6 your document?

7 DR. MARTY: It's not in writing, and it's not
8 included in the document.

9 DR. BLANC: Well, I would say that any member
10 of the public who opens up the document and suddenly
11 sees that there are not pesticides and no --
12 particularly no acetylcholinesterase inhibitors
13 included.

14 In fact, I would say that based on a narrow
15 reading of the statute -- first of all, I don't
16 necessarily agree with that interpretation based on what
17 I've read, but I would say even if that was correct in
18 the narrow sense, isn't it also true that it refers only
19 to -- the line that must have been interpreted in that
20 way refers to pesticides in their pesticidal uses.

21 So if there was any cholinesterase inhibitor,
22 let's say, that was ever used for any reason that was
23 not pesticidal and if it would be combined with the
24 effects of exposures that wouldn't fall under your
25 statute, you're supposed to consider that too as a

1 cumulative issue.

2 DR. MARTY: I can't really answer that. You
3 know, all I can say is what the attorneys have told me
4 is that this statute does not apply to the TACs that are
5 identified by DPR's director.

6 DR. BLANC: And yet your text says, "We looked
7 at all TACs." It doesn't say we looked at all TACs
8 except those TACs which involve pesticides.

9 DR. MARTY: It's because we actually did, but
10 as the process evolved realized we couldn't handle the
11 pesticides under this statute.

12 CHAIRMAN FROINES: I think that the problem
13 with, also, I think what Paul's raising in part, is that
14 if you have a chemical manufacturing company that makes
15 pesticides, then they would fall under this statute and
16 should not be excluded.

17 DR. MARTY: The other issue is that there are
18 not very much data on ambient concentrations of
19 pesticides. So within the paradigm we used, it's not
20 particularly easy to deal with the exposure aspect for
21 the pesticides, but there are -- you know, I can't argue
22 the law because I'm not a lawyer, but this is just what
23 we've been told. We can't.

24 DR. ALEXEEFF: George Alexeeff here with OEHHA.
25 If you look on page A-12, this is where we're actually

1 quoting. We have a copy of the statute in here.

2 CHAIRMAN FROINES: Yes.

3 DR. ALEXEEFF: And go down to subsection D;
4 okay? And now we're in the part about the listing, just
5 the listing part, what we're talking about today and
6 putting things on this children's related list. It
7 says, "Toxic air contaminants evaluated and listed
8 pursuant to this section shall not include substances in
9 those uses that are not subject to regulation by the
10 state board pursuant to this chapter."

11 So it does refer to, in part, what you were
12 just saying about the pesticidal use, but, basically,
13 we're restricted to those uses which the Air Board can
14 regulate. Now, we can make that clearer in the
15 document.

16 CHAIRMAN FROINES: But, Paul -- I mean, pardon
17 me, George, we identified ethylene dibromide as a TAC,
18 not through the Air Board but through the -- not through
19 the DPR but through the Air Board.

20 DR. ALEXEEFF: Correct.

21 CHAIRMAN FROINES: So that, in fact, there are
22 chemicals that are used or produced or formulated which
23 are pesticides but are -- which would then fall under
24 that designation of the state board.

25 DR. ALEXEEFF: Correct. Correct. And we tried

1 to retain those. So we could try to clarify that as
2 well in the list, which ones fell out, because of -- to
3 our knowledge, they were only emitted in their
4 pesticidal use.

5 DR. MARTY: Okay. I did want to --

6 CHAIRMAN FROINES: I think it's important to
7 stress for everybody in the audience and on the panel
8 that this is the first time any state or agency or
9 federal government has attempted to identify compounds
10 on the basis of their differential susceptibility, so we
11 are -- this is going to be under a real microscope, so
12 we really want to be sure to do it as well as we can.
13 So I think this is -- everybody should be prepared.
14 This is going to be a long day.

15 DR. MARTY: Okay. I did want to --

16 DR. GLANTZ: Especially for Melanie.

17 DR. MARTY: I did want to point out some of the
18 chemicals that we put back on the list, even though they
19 didn't make these initial cuts asbestos, a carcinogen
20 with a very long latency; carbon disulfide, we're
21 concerned about neuro and repro developmental toxcs;
22 glycol ethers, which are known developmental toxicants;
23 and hexane, we didn't have good data in terms of ambient
24 concentrations, but there are several large sources of
25 hexane in the state that are stationary sources, and

1 it's a peripheral nervous system intoxicant.

2 Isocyanates, there are -- we don't have good
3 ambient concentration data. There are a number of
4 sources -- stationary sources of isocyanates in this
5 state. They're potent sensitizers, so we're concerned
6 about those from an immuno-toxic perspective.

7 Mercury, we didn't have good data, but mercury
8 is a well-known developmental neurotoxicant. There is
9 widespread exposure in California. Although, it's
10 largely -- it's not necessarily from mercury that was
11 initially airborne.

12 And then we actually also added back in ethyl
13 ketone because of widespread emissions and potential for
14 increased use in consumer products because U.S. EPA may
15 list -- delist it as an ozone reactive volatile organic.

16 DR. ATKINSON: So you mentioned hexane coming
17 back in. Hexane is just one of many gasoline
18 ingredients, so anything else that's got as much
19 toxicity as hexane would probably be -- you'd get about
20 as much exposure, depending on how much is in the
21 gasoline. Even if there's no --

22 DR. MARTY: Yeah. Most of the important
23 chemicals in gasoline -- important in terms of we know
24 what -- something about their toxicity, did actually end
25 up in the final 35 that we did literature reviews on.

1 CHAIRMAN FROINES: We don't have that list.

2 DR. MARTY: You don't. I'm sorry. I meant to

3 put it as a slide and I didn't.

4 DR. GLANTZ: Yeah, we do. The 35? That's

5 Table D.

6 DR. MARTY: Oh. But I think what John means is

7 we don't have the list that I just rattled off.

8 DR. GLANTZ: Yeah. Well, I was going to

9 suggest that when you get the transcript, you can copy

10 it into the document. That would be very helpful.

11 DR. MARTY: Okay.

12 DR. GLANTZ: I mean, this gets back to really

13 making it -- I mean, I think the reasons that you

14 stated, Melanie, were very reasonable, and I think you

15 need to state those in the document for people to see

16 that's why you did it. It wasn't arbitrary. Those

17 are -- I think what you said is very sensible. It just

18 needs to be said in the document.

19 DR. MARTY: Okay. Table C, which is in front

20 of you, shows the 95 that did make it past this first

21 cut.

22 DR. BLANC: Table which? I'm sorry.

23 DR. MARTY: C. And it's alphabetical order.

24 It's not an indication of the ranking. You also have

25 Table 1, which is the ranking, which I think all of you

1 have already seen because we sent it to the panel with
2 the document.

3 But I do want to point out that the
4 quantitative ranking we did based on ambient
5 concentration data, reference exposure levels and unit
6 risk factors has limited utility in terms of
7 prioritizing for TACs that may impact children. The
8 health criterion aren't necessarily developed around an
9 endpoint that may impact children.

10 So it's -- you're dealing with existing
11 information. There's lots of newer information in the
12 literature that we needed to look at, which is why we
13 did the focus literature reviews.

14 Andy, could I have the next slide?

15 DR. GLANTZ: Now, the order in Table 1 that you
16 gave us now, these are ordered by the chronic -- the air
17 concentration over the REL and the risk. You took that
18 and then you took -- so you took the air concentration
19 over the REL and the unit risk times the air
20 concentration, and those are the last two columns.

21 DR. MARTY: Yes.

22 DR. GLANTZ: And then I'm just trying to make
23 sure I understand this. And then you sorted the list
24 based on the air concentration over the REL, and then
25 you went down and looked at the risk times the air

1 concentration, and if you had something that the first
2 sort seemed to put in the wrong place, you then moved --
3 you applied judgment to move it up or down.

4 DR. MARTY: Right.

5 DR. GLANTZ: To sort of balance the two
6 different outcomes.

7 DR. MARTY: Right. And --

8 DR. GLANTZ: So that's how you ended up with
9 this -- with this list, and these are not alphabetical
10 order. These are in the order of this --

11 DR. MARTY: They're pretty much in the order by
12 the air concentration over REL. They're not necessarily
13 in order by cancer risk.

14 DR. BLANC: Which list now? I'm sorry.

15 DR. MARTY: This is Table 1. It's the list
16 that has the --

17 DR. BLANC: Okay.

18 DR. GLANTZ: And that's the 95 then; right?

19 DR. MARTY: Yes. There's actually some that
20 didn't -- that ended up in the 95 that are not scored
21 here due to lack of ambient concentration data.

22 DR. GLANTZ: And then can you tell us -- when
23 you did the air concentration, you sorted by air
24 concentration over REL. Can you through and tell us
25 which ones you put in a different place than that order

1 based on the cancer data?

2 DR. FRIEDMAN: That's what I was asking before,
3 and you said we should defer it --

4 DR. GLANTZ: Right. I know.

5 DR. FRIEDMAN: -- until she was done.

6 DR. GLANTZ: All right. I'll be quiet.

7 CHAIRMAN FROINES: Melanie, I'm confused about
8 something. Your Table D has a list of 35 chemicals, and
9 this document that Stan and Paul were just talking about
10 has 88 on it.

11 And, for example, you have
12 N-Nitrosodimethylamine, which has a cancer risk times
13 air concentration of 1.2 times 10 to the minus 2, which
14 is the second highest number in that order, and yet it
15 doesn't make the list of 35. Can you say why? Because
16 clearly if you asked the question from the point of view
17 of carcinogenesis, it would be a high player, a
18 significant compound. And the same with dimethyl
19 sulfate, although, I don't believe those exposures --

20 DR. MARTY: Okay. The ambient air
21 concentration data was of variable quality. We had a
22 lot of confidence in the stuff we got from ARB. They
23 also had data that they collected from around the U.S.
24 primarily that we had less confidence in, and, in some
25 cases, it was just one -- a single measurement. The

1 nitrosamine data, we didn't have a lot of confidence in
2 the air concentrations. It rang bells for us for
3 certain.

4 We had more confidence in some of the other
5 chemicals from a toxicological perspective in terms of
6 differential sensitivity, and we had more confidence in
7 some of the other chemicals from the perspective of
8 quality of the ambient concentration data.

9 CHAIRMAN FROINES: But if you have something
10 that is -- if you have two compounds that are two orders
11 of magnitude greater in their cancer risk than
12 everything else, one could say why would you exclude
13 them?

14 DR. MARTY: Well, the only argument you would
15 make to include them was that you were concerned that
16 because they were carcinogens there's automatically a
17 differential impact in children. We are evaluating
18 that, the issue of age and exposure and weighing potency
19 for age and exposure, but we're not there yet. Our
20 methods for doing that are not ready for prime time.

21 So we were -- while it is a factor and it is a
22 concern. It's not necessarily enough to bump other
23 chemicals out of the way.

24 DR. GLANTZ: Well, I --

25 CHAIRMAN FROINES: It's not clear why it

1 doesn't make the list of 35, which would then be 26.

2 DR. GLANTZ: If I just before -- can I go back
3 one step?

4 CHAIRMAN FROINES: Let me just finish this
5 because -- let me just finish this train of thought.
6 Let me say one -- let me say two things.
7 N-Nitrosodimethylamine is a product of oxidation of
8 unsymmetrical dimethylhydrazine. It's found at
9 Rocketdyne. You find it at that air force -- that
10 Aerojet in Sacramento. It is a product of places where
11 hydrazines have been used. So we know it exists in
12 California, at least as a residual from those past uses.

13 There are probably 2,000 papers in the
14 literature on the carcinogenicity of
15 dimethylnitrosamine, so that you have an enormous
16 database. You actually have evidence of exposure. So
17 it seems to me that -- I don't understand how you could
18 then say, "We don't want this on our list of 35 to
19 evaluate." I mean, there is probably no compound that
20 has as many publications on carcinogenicity as that
21 particular compound.

22 So one could look at it from the standpoint of
23 differential susceptibility. So it doesn't make any --
24 I don't understand it, and I raise it only because the
25 issue of everybody's understanding of why things are

1 where they are is really important as everybody has been
2 saying.

3 DR. ATKINSON: It's largely there, I assume,
4 because -- in that position in that table because of the
5 ambient air concentration data concentrated points.

6 DR. MARTY: That's the problem.

7 DR. ATKINSON: It looks sort of high to me.

8 CHAIRMAN FROINES: It's very high.

9 DR. ATKINSON: This stuff photolyzes with a
10 lifetime of about 5 minutes, so here in the daytime you
11 wouldn't expect it.

12 DR. MARTY: That's precisely the problem we
13 had. We had not very much confidence in that ambient
14 air concentration data.

15 DR. BLANC: But, again, doesn't your statute
16 address potential air exposure as well as measured air
17 exposure? And, therefore, isn't the technology --

18 DR. MARTY: What it says is "consider public
19 exposures to the toxic air contaminants." I don't think
20 it's prescriptive in how you do that.

21 DR. BLANC: But you have interpreted it as
22 being prescriptive because you said if we don't --

23 DR. MARTY: Not really.

24 DR. BLANC: Haven't you said if we don't have
25 air monitoring level data showing it's there, or we

1 don't have toxic inventory release data, even though we
2 have reason to believe from other logical analyses that
3 it is there, that --

4 DR. MARTY: We have started with the best data
5 available, which is the ambient air concentration data
6 from ARB. We added in other data that we had, some of
7 it of varying quality. We looked at the emissions
8 inventories from stationary and mobile sources. All of
9 those things fed into the decision of whether or not
10 there's exposure and whether the exposure is
11 significant. It's not a process that is without flaws.

12 CHAIRMAN FROINES: I think the problem -- I
13 think the generic problem -- and this happens at EPA.
14 It happens with all agencies that deal with regulation
15 as well as science, and that is that they tend to chase
16 their tails. They tend to pursue chemicals that are
17 regulated, and then they pursue those chemicals further
18 then they pursue those chemicals further and
19 N-Nitrosodimethylamine never gets into the loop because
20 it's not a regulated chemical.

21 So the problem is that you keep looking at the
22 same compounds repeatedly. And I think the danger is
23 that there needs to be a way in which other chemicals
24 can enter into the evaluation process because they may
25 represent problems that are as yet unidentified or

1 having not been pursued.

2 And I think that's what Paul's raising about

3 the manganese question because I think -- Roger is

4 right. N-Nitrosodimethylamine is probably a problem at

5 Edwards Air Force Base and Aerojet and at Rocketdyne,

6 but it's not a problem anyplace else. It is a

7 historical problem from the use of a particular

8 hydrazine, but it still has 2,000 papers on its

9 carcinogenicity.

10 And so if it can never make its way into the

11 process, then we never think about it. We keep looking

12 at the ones we already know are problems, and manganese

13 is another example of that kind of issue.

14 DR. ATKINSON: But I thought it was also in the

15 cigarette smoke, the --

16 MS. REPORTER: I'm sorry. Could you speak into

17 the microphone?

18 DR. ATKINSON: -- N-Nitrosodimethylamine.

19 CHAIRMAN FROINES: Absolutely. In fact,

20 they're in large quantities.

21 DR. ATKINSON: Also the exposure.

22 CHAIRMAN FROINES: Yeah. That's right. And I

23 don't know about nitrosamines from diesel, do you?

24 DR. ATKINSON: What?

25 CHAIRMAN FROINES: Do you know about

1 nitrosamines from diesel or gasoline?

2 DR. ATKINSON: I shouldn't -- I wouldn't expect
3 to find them. The other place you might find them is
4 from cattle feedlots.

5 CHAIRMAN FROINES: Um-hmm.

6 DR. ATKINSON: Oxidation of a means.

7 DR. BLANC: Melanie, can I ask another
8 clarification of methods?

9 DR. MARTY: Sure.

10 DR. BLANC: So going from Table C to Table B
11 and then to -- from Table B to Table C -- I'm sorry --
12 and then from Table C to your list of 35, when there
13 are --

14 DR. MARTY: That's the next slide.

15 DR. BLANC: Yeah. On -- it's just a methods
16 question. On Table C, there are, in fact, pesticidal
17 chemicals, so the exclusion of pesticides occurred at a
18 later stage for some, or are these pesticides for which
19 the ARB has already --

20 DR. MARTY: It was really when we were going
21 from the 95 to picking which ones we wanted to do focus
22 literature reviews on that we realized we couldn't look
23 at pesticides in their pesticidal use.

24 Now, acrolein is used as an herbicide, but it's
25 also a product of incomplete combustion.

1 DR. BLANC: Right.

2 DR. MARTY: So looking at acrolein --

3 DR. BLANC: Was okay.

4 DR. MARTY: -- was okay.

5 DR. BLANC: So going back to my earlier

6 question about parathion, which was really a question

7 about organophosphates, were there no organophosphates

8 at all that made it to Table C therefore? And obviously

9 not -- and that would only be on an exposure reason

10 because you had not yet --

11 DR. MARTY: Yes. That's right. It would be on

12 the exposure reason.

13 DR. BLANC: So the only ones for which there

14 were no organophosphates already listed as TACs for

15 which there would be any ambient exposure to any one of

16 them because clearly you would have to consider the

17 combined effects?

18 DR. MARTY: Could you -- I'm not sure what

19 you're asking.

20 DR. BLANC: I'm asking again for transparency.

21 Let's say I'm reading this down the line, and I get to

22 Table C, and then I -- and then there's a footnote, not

23 there currently, which says, "This list includes

24 pesticidal chemicals with TACs that are pesticides and

25 have no other use whatsoever; and, therefore, the ARB is

1 excluded from regulating them, and the statute excludes
2 us from looking at them. And so at this point, although
3 we would have looked at them, if we could have from a
4 scientific point of view, from a regulatory point of
5 view we're prohibited."

6 DR. MARTY: We could put that footnote in.

7 DR. BLANC: I would put it there in caps and
8 bold.

9 DR. MARTY: I don't think it's that simple.

10 DR. BLANC: Why isn't it that simple?

11 DR. MARTY: Because we still aren't at the
12 point where we have scientific evidence for children,
13 either on an individual basis or population-wide basis,
14 being impacted more than adults. So while that's true
15 and we can put that footnote in, it's just one piece of
16 the puzzle. It's not the whole reason, perhaps, that
17 certain things were not looked at.

18 In the case of pesticides, we really can't --

19 DR. BLANC: I'm talking about -- okay. I'm
20 talking about acetylcholinesterase inhibitors.

21 DR. MARTY: Inhibitors. There's not much we
22 can do because of the way the statute is written.

23 DR. BLANC: I understand that, but let's say
24 you didn't have that statutory prohibition. Just as a
25 scientist and a public health regulator, wouldn't you

1 have been very interested in organophosphates or any
2 acetylcholinesterase inhibitors because of previous
3 discussions in terms of pediatric issues in
4 acetylcholinesterase functions?

5 DR. MARTY: Yes.

6 DR. BLANC: So from a scientific point of view,
7 wouldn't anything that was an acetylcholinesterase
8 inhibitor have been of particular interest in this
9 process had it not been specifically prohibited from you
10 looking at it?

11 DR. MARTY: We would have been interested in
12 it, yes. I --

13 DR. BLANC: Okay.

14 DR. MARTY: You folks had a presentation on
15 OPs, and the potential for differential impacts comes up
16 when you're looking at tyrosinase, for example.

17 DR. BLANC: So don't you think that one
18 potential utility of your document in terms of public
19 health protection would be to highlight areas for which
20 the science will direct you to look but for which your
21 hands are tied from a regulatory point of view?

22 DR. MARTY: We could put that in there. Yes.

23 CHAIRMAN FROINES: I think it's -- I think what
24 Paul is raising is that this is an extremely important
25 document, and if one views it narrowly, one comes out

1 with a list of five chemicals. And I think, for the
2 record, for OEHHA to be on record of defining the
3 breadth of the issues is really very important because
4 it forms the basis for subsequent legislation or
5 activities that might take you to another level of
6 investigation.

7 DR. MARTY: The light just came on. I'm sorry.
8 Yes. If you're viewing a document as not with my brain
9 but a brain of an outsider, you would want to know why
10 pesticides weren't in there.

11 DR. BLANC: Well, I'm speaking now specifically
12 about organophosphates. We could discuss organochlorine
13 compounds separately, and I think the science is
14 probably more complicated.

15 DR. MARTY: Yes.

16 DR. BLANC: But --

17 DR. MARTY: This document has many audiences,
18 in other words.

19 DR. GLANTZ: Well, can I --

20 CHAIRMAN FROINES: Yeah, Stan. We cut him off.

21 DR. GLANTZ: I'd like to go back to Gary's
22 question now, which now we've reached the precisely
23 right time to ask. If you look at table -- I'm just
24 trying to understand and get on the record exactly what
25 you did. And so we have Table 1, which is a list --

1 which is what? That's list C; right? No.

2 DR. MARTY: List C is the 95 --

3 DR. GLANTZ: Okay.

4 DR. MARTY: -- TACs for which there are --

5 DR. GLANTZ: Okay. And that's what ends up in

6 Table 1; right?

7 DR. MARTY: Pretty much with a few exceptions

8 where we couldn't rank them because we didn't have --

9 DR. GLANTZ: Okay. So while Paul was talking,

10 I went through Table 1 and --

11 DR. BLANC: You mean you weren't listening to

12 me?

13 DR. GLANTZ: I was listening. I can do two

14 things at once.

15 Anyway, I just went through Table 1, and you're

16 right. For the most part things are ranked by the air

17 concentration over the REL. But let's just go down to

18 ones that aren't, and you can just briefly tell us why

19 you put them where you put them; okay? I can tell you

20 it's No. 6, 7, 10, 21, 31, 32 and 33, 41 and 42, 46, 58,

21 and then there's all the stuff at the bottom.

22 So I think it would be instructive just if you

23 could briefly just tell us -- because all the ones that

24 are ranked by the RELs, that's obvious what you did. So

25 if you could just go down and say why did you put, you

1 know, the things where you put them on the list.

2 DR. MARTY: Okay. We can do that, but I want

3 to caveat it by saying that this breaking has limited

4 utility in coming up with five TACs that may cause

5 infants and children to be especially susceptible.

6 DR. GLANTZ: Okay. We're just trying to

7 understand the process.

8 CHAIRMAN FROINES: I don't think you should go

9 through that entire list.

10 DR. GLANTZ: Okay.

11 CHAIRMAN FROINES: I think if Stan wants more,

12 he can ask for more, but let's do it -- I'm worried that

13 we probably should --

14 DR. MARTY: Okay.

15 CHAIRMAN FROINES: -- get to the chemicals

16 after lunch, so that between now and lunch we want to

17 deal with the methodology which gives us about an hour

18 to do that.

19 DR. FRIEDMAN: How about No. 21?

20 DR. MARTY: Okay. But --

21 DR. FRIEDMAN: Which, you know --

22 DR. GLANTZ: Pick a couple. I'd like to hear

23 about a couple.

24 DR. MARTY: For one thing, No. 10, betadine

25 made the final cut for us to look at differential

1 impacts. So where it is with respect to 8 times 10
2 minus 5 being a bigger number than 2 times 10 minus 5
3 doesn't really matter in the final analysis. We stuck
4 it on the list of 35.

5 DR. GLANTZ: Okay.

6 DR. MARTY: Dimethyl sulfate and dimethylane --

7 DR. GLANTZ: Wait. Wait. Wait. But with
8 butadiene -- so you're saying that -- so tell me again
9 why? I'm just trying to go through the process. So why
10 did you put it where you did? And I realize in the end
11 the lists end up back to being alphabetical. But why?
12 It ended up pretty high on the list, you know. I'm not
13 asking, like, why is it No. 10 instead of No. 9 or
14 No. 11, but why did you push it up? Because if you look
15 at the REL, it would have been way down. It would have
16 been like 20, 25 or something.

17 DR. MARTY: Right.

18 DR. GLANTZ: So why did you put it about where
19 you put it?

20 DR. MARTY: Because of the potential for
21 carcinogenicity and widespread exposure.

22 DR. GLANTZ: And then what about -- you talked
23 about No. 7; right? That was what John was talking
24 about.

25 DR. MARTY: Yes. And dimethyl sulfate falls in

1 there also.

2 DR. GLANTZ: Okay.

3 DR. MARTY: We were pretty unsure of those

4 ambient concentrations in that. I probably should have

5 just taken them out entirely, but I didn't.

6 DR. GLANTZ: Well, but no. But I'm asking --

7 so you -- those -- if you look at the cancer risk as

8 computed, those are like really huge numbers, so that's

9 why you pushed those up there. Okay. And then Gary had

10 asked about chromium.

11 DR. FRIEDMAN: You base that on the cancer

12 number it looks like.

13 DR. MARTY: Yes.

14 DR. GLANTZ: Why did you put it where you did

15 in the -- because all the other cancer numbers on there

16 were like 10 to the minus 5, is that why you put that

17 there?

18 DR. MARTY: Right.

19 DR. GLANTZ: But then if you go down to, like,

20 31, 32, 33 you've got a bunch of, like, 10 to the minus

21 5 cancer numbers. How come you didn't put those higher?

22 DR. MARTY: We weren't particularly concerned

23 about chlordane and heptachlor which are banned

24 pesticides. And tetrachloroethane, I can't remember why

25 we didn't move it up. For one thing, it's a Class 3

1 carcinogen, an IARC 3, on the U.S. EPAC, so that you'd
2 be less worried about a 3 or a C than something like
3 perc, which is a 2A, or something like beryllium, which
4 is a 1.

5 DR. FRIEDMAN: So even though it's a lot of
6 work, I would recommend that you just be explicit in the
7 document about these decisions and how you arrived at
8 it.

9 CHAIRMAN FROINES: Melanie, I have a question.
10 Continuing this list of -- you know, I made the point
11 about chemicals that get regulated get continually
12 looked at, and chemicals that aren't don't get looked at
13 very effectively by these processes, and I'll give you
14 an example of one that I think is extremely important
15 that is on this list ranked 82nd, and that is
16 naphthalene.

17 And George knows that's a compound of
18 particular interest to me for two reasons: One because
19 the chronic (phonetic) amobioassays are positive in both
20 rats and mice at this point. So one would probably --
21 even though it hasn't been necessarily ranked by
22 international agencies like IARC, it's still one that
23 NTP would consider a carcinogen. And I don't know what
24 ranking it would have, but we would have to take it
25 seriously.

1 And then if you look at the data that Roger and
2 Janet developed in the -- when they were looking at PAH
3 concentrations, it's certainly in very high
4 concentration in California. And so how a compound of
5 that magnitude -- of that importance ends up at 82nd is
6 a mystery to me.

7 DR. MARTY: Well, it's actually -- since it's a
8 polycyclic aromatic hydrocarbon and we decided to look
9 into polycyclic aromatic hydrocarbons because there's a
10 lot of information on developmental toxicity and
11 potential or differential effects, it's actually
12 included under the PAH.

13 CHAIRMAN FROINES: It's included under the PAH,
14 and that also is something -- this is a side bar, so I
15 won't pursue it, but it's something that worries me
16 because
17 we -- this panel put a lot of time into PAHs some years
18 ago and identified them as TACs, and there has been no
19 single, control-strategy approach taken, even though we
20 found it a TAC. So that in some cases I prefer that we
21 look at individual compounds to try and drive the system
22 to some extent because that's out of the risk assessment
23 mandate for this panel.

24 But the point is that sometimes it's useful to
25 look at chemicals and not simply lump them because

1 lumping them may end up the fact that they get lost in
2 the shuffle if we're not careful. And naphthalene I
3 think is a chemical that definitely should not be lost
4 in the shuffle.

5 DR. MARTY: The only thing I can say is that
6 PAHs are again being evaluated under the TAC program.
7 ARB has requested us to review the information on PAHs,
8 naphthalene among them. So it will be addressed through
9 that program.

10 DR. BLANC: So in terms of on the table that
11 this relative ranking which then derives very closely to
12 the ones that -- the 35 that end up on Table D is
13 closely driven by this table with certain exceptions.
14 This has a big impact. Again, these are questions
15 trying to understand the process you used. So somehow
16 table -- this table --

17 DR. MARTY: Could we have the next slide, and I
18 can talk about that? It is -- it does drive it
19 somewhat.

20 DR. BLANC: It is one of the factors.

21 DR. MARTY: It's one of the factors.

22 DR. BLANC: So that if something --

23 DR. MARTY: But the disconnect comes in. If
24 you just look at the top 50 or 60 by rank, you may not
25 be picking up chemicals for which you know there's a

1 differential impact.

2 DR. BLANC: Right. And then things that didn't

3 appear on the table were all -- weren't necessarily

4 excluded?

5 DR. MARTY: Right.

6 DR. BLANC: And those are some eight in number?

7 DR. MARTY: Right.

8 DR. BLANC: And those don't appear here because

9 you don't have the draft or an adopted REL example? Is

10 that an absolute reason?

11 DR. MARTY: It's primarily because we didn't

12 have good ambient concentration data to use in the

13 ranking.

14 DR. BLANC: Well, you have lots of things

15 without ambient concentration data here.

16 DR. MARTY: Right.

17 DR. ATKINSON: In fact, it looks to me as

18 though that would have been somewhere to put a fair

19 amount of effort into, going through the literature and

20 trying to find out at least some idea. Admittedly, it's

21 going to be time and place dependent, but at least get

22 some idea of what sort of concentrations are out there

23 and ambient.

24 DR. BLANC: So it can't be --

25 DR. MARTY: We did do that --

1 DR. ATKINSON: That's what drives the whole
2 thing.

3 DR. MARTY: We did do that for certain
4 chemicals that we had concerns about, but we could not
5 possibly do that for all 200 chemicals given the time
6 frame.

7 DR. BLANC: No. But, Melanie, I'm just trying
8 to understand this. I mean, half of these don't have
9 air concentrations to REL levels --

10 DR. MARTY: Okay.

11 DR. BLANC: So that can't be the reason why
12 some of these aren't on that table.

13 DR. ATKINSON: Especially those with --

14 DR. MARTY: Okay.

15 DR. ATKINSON: -- small RELs.

16 DR. MARTY: Your first assumption was correct.
17 Dave is correcting me. It's because they didn't have
18 health criteria, either a developed REL or being a risk
19 factor. In one case --

20 DR. BLANC: Either draft or --

21 DR. MARTY: In one case, which I need to get on
22 the record, lead was initially going to be dealt with
23 under SB 25 in the criteria air pollutant process, which
24 is a separate process. It was decided that they weren't
25 going to deal with it under the criteria pollutant

1 process. They wanted me to deal with it under the toxic
2 air contaminant portion of the statute. So lead is --
3 gets added in partway through the process.

4 DR. BLANC: And the other ones are for which
5 there's neither a draft nor an accepted REL?

6 DR. MARTY: Yes. Staff is saying yes.

7 DR. BLANC: Can you list those so that they're
8 in the transcript?

9 DR. MARTY: Those were the ones that I went
10 through a few minutes ago. Okay. It's -- MEK was one
11 and CS2 was one, but some of these others, it must have
12 been -- it must have been the concentration data that
13 made us add it back in.

14 These are the ones that have ambient data.
15 He's asking for ones that didn't have RELs. We've just
16 got to go back and list out which ones had RELs and
17 which ones didn't have RELs. I can't do it right here.

18 DR. GLANTZ: Let me ask a question. Are you
19 having fun now?

20 DR. MARTY: No.

21 DR. GLANTZ: Okay. If you go down to No. 59 in
22 the last part of the list, those are the ones where
23 there's like nothing in the last two columns of the
24 paper. Okay. Do you have anything that you just want
25 to say about that? Is there any comment, you know, to

1 explain sort of -- how did they even get into this
2 table, if there's, like, nothing there? For those,
3 there's no ambient -- I guess are those ones where
4 there's no ambient air concentration data, but you think
5 they're bad?

6 DR. MARTY: And we had emissions inventory
7 data.

8 DR. GLANTZ: I see.

9 DR. ATKINSON: I mean, I'll just take one
10 example, ammonia, which is 64. It's got this rather
11 large REL of 200, but if you go out to Mira Loma where
12 ARB conducted a study two or three years ago, they were
13 seeing up to 700 ppb out of the feedlots. So even with
14 a huge REL like that, you can still end up with a fairly
15 decent-sized number regarding the air concentration
16 amount.

17 DR. MARTY: Okay. Fifty-nine on, there's no
18 ambient data, but there were tox data.

19 Ammonia is on the 95 TACs that we chose a
20 portion of to do focus literature searches. One of the
21 reasons is there's -- obviously, there's a lot of
22 exposure to ammonia. It's used tremendously. There's
23 huge emissions from stationary source.

24 DR. GLANTZ: Okay. Well, then -- so the next
25 question and then you could put your next slide up.

1 We'll try to keep your frustration --

2 DR. MARTY: Go back.

3 DR. GLANTZ: To the next slide. That's the

4 one.

5 DR. MARTY: This is going from the 95 to the 35.

6 DR. GLANTZ: Okay. Now, I want to ask a

7 question about that.

8 DR. MARTY: Can I go through the slide first.

9 DR. GLANTZ: Okay.

10 CHAIRMAN FROINES: Go ahead.

11 DR. GLANTZ: Go to the next slide.

12 DR. MARTY: Because we had limited resources

13 and time, the deadline of the statute, we couldn't

14 possibly do a focus literature search on all 95, so we

15 decided to take about a third of them and look at about

16 a third of them.

17 We focused on some that had -- that ranked high

18 because of the REL and the ambient, for example,

19 acrolein. We focused on some that ranked high because

20 of the carcinogenicity hoping to find something that may

21 shed light on whether there was differential

22 sensitivity.

23 But we also ended up weighting those with known

24 toxicological properties that have been shown or might

25 be expected to demonstrate differential sensitivity in

1 young persons or mature animals.

2 For example, lead and mercury are well-known
3 developmental toxicants. Despite the fact that there
4 aren't huge exposures on a regional basis, we had
5 concern of those -- over those for the toxicology
6 information and the epidemiology information that's out
7 there, and there actually are hot spots facility
8 emissions of those two chemicals out there.

9 DR. GLANTZ: Okay. I think that's all
10 sensible. The technical question I have -- and, again,
11 this is just trying to get everything out there on the
12 record. It's not that I think you're a bad person.

13 So if you take the 95, if you take Table 1,
14 which has a ranking that we now more or less understand,
15 how does Table C -- if you took the top 35 compounds on
16 Table 1, okay, and you're saying to us, for reasons
17 which I personally think are quite reasonable, that you
18 didn't slavishly follow this list, how did -- what is
19 there on Table C which is different from the top 35 --
20 or not Table C.

21 DR. MARTY: Table D has the --

22 DR. GLANTZ: Table D. I'm sorry. What is
23 there on Table D that is different from the top 35
24 chemicals on Table 1 and why? If you could just go
25 through and explain to us those that were added or

1 deleted from the top 35 of Table 1. You've already
2 dealt with a couple of them.

3 DR. MARTY: Okay. Acrolein is No. 1, so that
4 made the cut. Acetaldehyde, it made the cut, and we're
5 concerned about the toxicity.

6 DR. GLANTZ: Okay. I'm just asking you just
7 the narrow question.

8 DR. MARTY: Okay.

9 DR. GLANTZ: If you take the top 35 in Table 1;
10 okay? What is there in Table D that isn't in the top 35
11 of Table 1, and why did you add it and then --

12 DR. MARTY: Okay. Asbestos got added in
13 because of concerns about long latency and shelf life of
14 kids. People who are exposed early in life to asbestos
15 end up with mesothelioma in their thirties and forties.

16 DR. GLANTZ: Okay.

17 DR. MARTY: So that was a concern.

18 Okay. I'm going from -- George is confusing
19 me. I'm going to try to talk about the things that
20 weren't in the top 35 that by this scoring -- they end
21 up in the top 35 on this scoring.

22 DR. GLANTZ: Yes. That's right

23 CHAIRMAN FROINES: I think that -- I think
24 that -- I think one has to establish criteria at each
25 level as a basis for the decision making and then that

1 drives how you then do it.

2 DR. MARTY: The --

3 CHAIRMAN FROINES: So I think that we don't
4 need to go through 35 different chemicals right now. I
5 think we need to describe what is the basis for the
6 differences between the first 35 in one table and the
7 next 35 in another.

8 DR. GLANTZ: And I think that-- no. Well, I
9 disagree with you. I don't think we need to go through
10 all 35, but I think Melanie has explained the criteria.
11 I'd just like her to very briefly just explain to us the
12 ones where they don't match up. So asbestos is one, and
13 there aren't that many of them.

14 DR. MARTY: Vinyl chloride is another. There's
15 not a lot of exposure on a regional-wide basis. There
16 are some concerns about hot spots exposures, for
17 example, measurable vinyl chloride levels near
18 landfills. So that's another reason. But if you look
19 at the toxicity piece, it's clearly more potent when
20 exposures occur either in utero or perinatally. So to
21 us, that was an important thing to get out and discuss
22 in this document.

23 DR. GLANTZ: Okay. What else? You already
24 mentioned lead and mercury.

25 DR. ATKINSON: Looks like dioxins.

1 DR. MARTY: Dioxins is another example where
2 there's a lot of toxicity information that indicate
3 differential effects. There's a lot of concern about
4 low level exposures to dioxins at current ambient levels
5 of exposure, and that's from all routes of exposure.

6 DR. ATKINSON: 1, 4-Dichlorobenzene, No. 40

7 DR. GLANTZ: And why was that?

8 DR. MARTY: I have to get back to you on that
9 one. I can't remember why we moved it up.

10 DR. ATKINSON: Well, that may be because the
11 pesticides fell out; right, things like chlordane and
12 heptachlor? So that would mean that the top 35 on
13 List D would have been the top 40 on List 1.

14 DR. MARTY: Yes.

15 DR. GLANTZ: So, basically, then -- so you
16 dropped out the pesticides, and then there are these
17 one, two, three, four, five, six you just discussed, and
18 everything else then would be in the top Tier on
19 Table 1; is that correct?

20 DR. BLANC: No. Carbon disulfide doesn't
21 appear on Table 1 because they didn't have a draft for
22 an accepted REL; is that correct?

23 DR. MARTY: Yes.

24 DR. GLANTZ: And then --

25 DR. BLANC: Lead wasn't there.

1 DR. MARTY: We were concerned about the
2 neuro-toxicant.

3 DR. GLANTZ: Okay. And then lead -- you
4 already talked about lead and mercury. Any others?

5 DR. MARTY: I'm remembering that we moved
6 benzopyrene up and actually the whole class of PHs, and
7 that again is driven by the information on the
8 toxicology of those compounds. It will be hard to
9 ignore that information because the exposures are lower.

10 DR. GLANTZ: There's lots of that in cigarette
11 smoke, too, actually.

12 CHAIRMAN FROINES: Well, I think we can --

13 DR. MARTY: The other issue is that --

14 CHAIRMAN FROINES: I really think this is
15 not -- that this reaches a level of usefulness. The
16 ultimate document has to describe the basis for decision
17 making so that everybody understands it. We don't need
18 to go through each one.

19 DR. GLANTZ: No. I think we've gotten this
20 adequately, and I think the reason that you're
21 presenting is all very fine.

22 But as John just said, I think this needs to be
23 spelled out in the document, and then I think nobody can
24 say, as several of the commenters said, "We don't
25 understand how you got the list." And I think people --

1 as somebody said, people can argue with you about the
2 judgment that was applied in getting the list and that's
3 their prerogative, but I think -- I think we have to
4 make it very, very clear how you ended up with this.

5 I think, again, from my meeting with Melanie
6 and her staff and what's been said today, I think they
7 employed reasonable criteria. I just think that they
8 were absolutely not explained, and I think that's what's
9 caused a lot of the difficulty.

10 So, anyway, so then you ended up with Table B.
11 Okay. And then we will now allow you to show one more
12 slide. So you did the focus literature reviews on all
13 35?

14 DR. MARTY: Yes. And some of them we have very
15 recently done the literature reviews.

16 DR. BLANC: Did you out source this?

17 DR. MARTY: Yes.

18 DR. BLANC: And is that -- that wasn't really
19 stated very explicitly in the document.

20 DR. GLANTZ: Well, they do that all the time,
21 though. I don't think that's an issue.

22 DR. BLANC: I think that is an issue because I
23 think that it could come back to be an issue, and I
24 think that transparency is very key. And there's
25 certainly nothing to be embarrassed about if you out

1 sourced it to, you know, reputable, you know, academic
2 bases, professional members.

3 DR. MARTY: We out sourced it to UCB, UCLA,
4 USC. What else? UCSF.

5 DR. FRIEDMAN: What did you out source? The
6 literature review and the summaries of the literature?

7 DR. MARTY: Right. We contracted the reviews
8 of most of the 35 out. Some of them were done in house.

9 DR. FRIEDMAN: But not the decision making.
10 Just the information gathering.

11 DR. MARTY: No. Just to get the information,
12 pull up the -- get us the papers, a summary of the
13 papers, then staff then took that information, read the
14 papers, decided whether we agreed or not with the
15 contractors, which in some cases we did not, and then
16 put the document together, choosing just those 11 that
17 we thought had the strongest information based on the
18 focus literature reviews.

19 DR. FRIEDMAN: I agree with Paul. I think
20 that's really helpful to know that, and it again does
21 not detract from your process.

22 DR. MARTY: Okay. We've never really put into
23 a document before whether we've contracted out or not.

24 DR. GLANTZ: I think that I actually don't
25 agree because I think that the document is OEHHA's

1 document, and I think they're the ones who are
2 responsible for what it says. And if they hired someone
3 to assist them in preparing it, then I don't see how
4 that's relevant.

5 DR. BLANC: I'll give you an example of how I
6 think it's relevant.

7 DR. GLANTZ: Okay.

8 DR. BLANC: I think it's relevant because if
9 you farmed out five chemicals to some whose area of
10 particular expertise was carcinogenesis, and you farmed
11 out five others to somebody whose area of research is
12 neurotoxicology, and those are chemicals both -- and you
13 very wisely put the five out that you have reason to
14 believe to act as neurotoxicants to researchers with
15 expertise in that area, that strengthens the conclusions
16 that you eventually drew. Unless, for some reason, you
17 have trepidations about how you did the out sourcing, it
18 would seem to be strength, not a weakness.

19 CHAIRMAN FROINES: I also think, quite frankly,
20 I agree with Paul. I'm on the Board of Scientific
21 Council of the National Toxicology Program, and all the
22 documents we get for review list the contracting
23 agencies that did the documents for NTP. So I think for
24 consistency it would be wise.

25 But also, I hate to say this, but some of those

1 documents that they have contracted for turn out not to
2 be very well done, and I think that the Board of
3 Scientific Council raises questions about the quality of
4 the documents. And so it seems to me that it's better
5 to have it all laid out on the table than to not have it
6 laid out. I think everybody needs to be able to
7 understand what went on in a process so that we can
8 improve the process. It's not --

9 DR. MARTY: We can put it in. We can put it
10 in.

11 DR. GLANTZ: Okay. I think the thing, though,
12 that's important to stress, though, I relent, but I
13 think the important thing to stress though is that they
14 may maintain someone on the outside to draft something
15 for them and collect information for them, but it's
16 their document, and I consider OEHHA to be the authors
17 of the document, even if they hire somebody to draft
18 something, because it is their -- I am assuming that if
19 something comes forward, it's OEHHA speaking, not some
20 contractor that they happened to hire. And I think
21 Melanie said that, that they took the material and then
22 they applied their professional judgment to what was
23 then forwarded to us.

24 DR. BLANC: That wasn't my implication.

25 DR. GLANTZ: Okay.

1 CHAIRMAN FROINES: But I think that the role as
2 envisioned by the legislature, this panel is a kind of
3 quality control. That's why we review what you do, and,
4 therefore, the more information we have about how you do
5 what you do, the better off we can fullfil our
6 responsibility.

7 I have a question. I think we could use a
8 five-minute break for the court reporter. We're also 25
9 minutes or so from -- how long do you think you're going
10 to go on, Melanie? I realize -- I realize that's a very
11 open-ended question.

12 DR. MARTY: Okay. I'm just looking at --

13 DR. GLANTZ: She has one more slide. Three
14 hours.

15 DR. MARTY: I have nine more slides. On the
16 process I only have, basically, two more slides, and
17 then I wanted to talk a little bit about one of the
18 endpoints that we chose as the basis for some of our
19 decisions and why we chose it.

20 CHAIRMAN FROINES: That's a very, very good
21 answer, and so we'll take a five-minute break for --
22 because closure is not within the --

23 DR. MARTY: Five minutes.

24 CHAIRMAN FROINES: -- immediate future.

25 (Recess.)

1 CHAIRMAN FROINES: Let's begin again.

2 DR. MARTY: Andy, can I have the next slide?

3 CHAIRMAN FROINES: Wait, Melanie. I don't

4 think we have everybody seated.

5 DR. BLANC: Here's Stan.

6 CHAIRMAN FROINES: Okay.

7 DR. MARTY: Okay. This slide points to the

8 criteria which we discussed in the document related to

9 what we were looking for in the focus literature reviews

10 of the 35 chemicals that we looked at. And the primary

11 thing was evidence indicating infants or children may be

12 more susceptible to the toxicity of that compound and

13 the strength of that evidence.

14 We also looked at nature and severity of the

15 effect. Particularly, is it an irreversible effect? Is

16 it something, for example, an eye irritation versus a

17 developmental defect? You would want to consider that.

18 We also looked at evidence that the existing

19 health criteria may be inadequate. Although, this

20 didn't play a large role in the final decision. And by

21 that I mean whether the existing reference exposure

22 levels for cancer potency factors would have adequately

23 protected children.

24 We also looked for potential difference in

25 susceptible to carcinogenesis based on either known or

1 plausible mechanisms.

2 We looked at the extent of exposure and/or the
3 magnitude of risk at ambient concentrations and
4 indications that infants and children might be more
5 heavily exposed to the materials, particularly, for
6 example, by deposition onto surfaces, which would occur
7 in the case of PAHs and others, dioxins, and that really
8 cuts to the issue of hand-to-mouth behavior in kids.

9 Next. We chose 11 chemicals or chemical
10 classes for potential candidates for listing based on
11 the information in the focus literature reviews. We
12 weighted heavily the known toxicological properties in
13 the compound. We weighted the extent of exposure,
14 strongly weighted evidence for differential toxicity.
15 Evidence of widespread exposure was also weighted and
16 then we -- within the 11, we propose a Tier 1 which
17 consisted of 5 chemicals for listing under the statute.

18 CHAIRMAN FROINES: There are two kinds of
19 exposures. There are exposures, for example, in the
20 ambient environment that are of consequence, and there
21 are differential exposure, namely, that a child who has
22 more outdoor time or what have you may have a
23 differential exposure. And so the extent of exposure is
24 really two categories, not one. So can you speak to
25 that issue?

1 DR. MARTY: Yes. There -- if you look at
2 something that has widespread exposure in terms of
3 regional, on a regional basis, PAHs or benzene, about
4 all you can say from the existing information is that
5 kids breathe more per unit body weight than adults.
6 They eat more, they drink more per unit body weight than
7 adults. So for those routes of exposures, kids will be
8 exposed to larger amounts of chemicals than adults given
9 everything else being equal. So in the same
10 environment.

11 CHAIRMAN FROINES: But things aren't equal.

12 DR. MARTY: Right.

13 CHAIRMAN FROINES: That's very important that
14 things are not equal.

15 DR. MARTY: That's right.

16 CHAIRMAN FROINES: So there has to be a
17 demonstration of differential exposure.

18 DR. MARTY: Well, what I just said doesn't help
19 you very much to figure out which chemicals are more
20 important from an aspect of differential exposure
21 because, essentially, kids will be more exposed to
22 everything, so it doesn't help you differentiate. We
23 did try to find some information --

24 CHAIRMAN FROINES: That's not really true. An
25 adult who drives two hours a day on the freeway behind a

1 diesel truck is going to have --

2 DR. MARTY: Yes.

3 CHAIRMAN FROINES: -- a higher exposure than

4 children playing in the yard.

5 DR. MARTY: I agree with that. What I meant is

6 given the same environment, if you stick them in a

7 chamber, the kids are going to breathe more. Give

8 everybody -- so within the same -- that's why it doesn't

9 help you very much to figure out what differential

10 exposures there are.

11 There's -- there are some data that can help

12 you, for example, time activity patterns to look at how

13 much time a kid spends in the car versus how much time

14 an adult spends in the car and so forth. Those types of

15 analyses are pretty time consuming and long, and we did

16 not do that for this initial prioritization.

17 We did, however, look for information in the

18 literature searches that brought those issues forward.

19 So, for example, there's some information for PAHs that

20 kids are more exposed to PAHs. There's lots of

21 information that kids are more exposed to lead,

22 primarily from hand-to-mouth behaviors.

23 So you are right. There's sort of the generic,

24 yes, you have ambient concentration data. That means

25 people are exposed. And then there's the more specific

1 exposure differences that are based primarily on time
2 activity patterns and behavior.

3 CHAIRMAN FROINES: So that means that the fact
4 that children have higher breathing rates and all those
5 other physiologic factors that you lay out in your
6 document, they were not used as a basis for defining the
7 chemicals that are on the lists in this document?

8 DR. MARTY: They really couldn't be used. All
9 you can say from that is kids have higher exposures to
10 everything. You can't say kids have higher exposures
11 to -- in the same environment, kids have higher
12 exposures, but you couldn't really use it to say one way
13 or the other unless you had specific information, like
14 for lead, for example, or for other chemicals where hand
15 to mouth is an important issue.

16 CHAIRMAN FROINES: But my point here is --
17 Stan, I'll get to you in a second.

18 My point is that you make -- in pages 3 through
19 9, you emphasize those physiologic differences, and the
20 problem in the document is that you then don't use that
21 area of emphasis for decision making. But it's never
22 made clear in the document that the differential
23 exposure based on physiologic characteristics was not
24 used for decision making, and I think that that's a
25 problem because of the nature of your emphasis when you

1 set out the differences between kids and adults.

2 So that you set it out, and then there's an
3 expectation on the part of the reader that it's going to
4 be used as a decision making basis, and then you don't
5 use it. And so that creates a problem for somebody
6 trying to read the document. It's hard to figure out --
7 it's hard to figure out what, in fact -- you know, who's
8 on first kind of.

9 DR. MARTY: Okay. You know, we did develop
10 that section on factors influencing why infants and
11 children might be more susceptible than adults, and the
12 whole purpose of that was to give a broad overview of
13 the types of factors that influence response to
14 toxicants. One of those is how much you're exposed. So
15 that's the reason while all that whole section is in
16 there.

17 It wasn't really meant to be applied to each
18 specific case in the back, but, you know, obviously that
19 wasn't clear, so we can just try to describe that a
20 little better.

21 DR. FRIEDMAN: Excuse me. Didn't you use a
22 difference like that, though, in selecting certain
23 chemicals in relation to asthma because of children
24 having smaller airways which are more easily blocked?

25 DR. MARTY: Yes. For -- yes, that's correct.

1 For physiologic differences. And, of course, there's --
2 I shouldn't say we didn't -- I shouldn't say we didn't
3 use them. That's not correct either. Certain things
4 applied in some of those chemicals. A lot of them
5 didn't, but it doesn't mean we didn't consider it or
6 think about it when we were going over one choice or the
7 other.

8 CHAIRMAN FROINES: But Gary's asking it --
9 Gary's asking a question which is very bothersome to me,
10 which is he's asking the question in a generic way. And
11 yes, of course, it's a given in a generic context that's
12 true. Children have smaller airways, therefore -- but
13 that doesn't mean that you then have any evidentiary
14 basis to show differential exposure.

15 DR. FRIEDMAN: Well, I guess what I was saying
16 is that we sort of came down with a blanket statement
17 that the material on 3 to 9 was not used at all. These
18 characteristics of children as comparison in adults was
19 not used at all in making the selection of
20 prioritization. What I was saying is that I think some
21 factors, particularly the narrowness of the airways,
22 this is an exception that that was used. That was the
23 point I was trying to make.

24 DR. ALEXEEFF: I think the point of -- George
25 Alexeeff of OEHHA. The point of the first section, we

1 did try to consider those factors. For example, on the
2 breathing rate information that -- a lot of -- most of
3 that information was in the CCAA document that we had on
4 exposures where we looked at activity patterns of
5 children and adults and built the exposure differences.

6 So we tried to look at that information to see
7 if it could be applied, and I guess there were specific
8 cases, as you mentioned, where we found some concerns.
9 Just asthma we looked at more carefully to see if there
10 was an issue that played out with the chemicals.

11 But some of the issues that Melanie pointed
12 out, for example, the overall breathing, breathing per
13 kilogram, okay, that was a factor that, in general,
14 children -- their whole distribution is -- indicates, in
15 general, they breathe more per kilogram body weight than
16 adults. So that wasn't a way of differentially choosing
17 any chemical.

18 We thought about -- we thought about it many
19 ways. You know, maybe we can look at particulates or
20 gaseous chemicals, but we couldn't come up with
21 something. What we could do is we could possibly add to
22 those sections whether or not that section led us to
23 something -- to a conclusion to list chemicals or to
24 identify chemicals or whether or not it was just a
25 general factor that we just, you know, sort of felt

1 might apply to all chemicals but not differentially to
2 every chemical.

3 DR. FRIEDMAN: That would be very helpful to
4 add statements like that.

5 CHAIRMAN FROINES: Stan.

6 DR. GLANTZ: I had a couple questions. I'm
7 still trying to get from Table D to 11. Poor Melanie.
8 The first question I have, which is just reflecting my
9 own ignorance, is in Table 1 of the document where you
10 list the 11, you have non-coplanar PCBs, and I don't see
11 that on Table D, or is that just they're called
12 something else?

13 DR. ATKINSON: They're coplanar PCBs.

14 DR. GLANTZ: Well, coplanar PCBs are in, but
15 they also list in Tier 2 "non-coplanar PCBs."

16 DR. MARTY: I'm looking for Table D. We had
17 lumped dioxins and PCBs together, and we should have put
18 coplanar and non-coplanar PCBs in that table.

19 DR. GLANTZ: That's two separate entries.

20 DR. MARTY: Right. This is the list of the
21 compounds that got literature reviews. It shouldn't
22 just say "coplanar PCBs" because we were looking at PCBs
23 generally. So if you scratched out the word
24 "coplanar" --

25 DR. GLANTZ: Okay. I think it would be -- this

1 is just, again, making it totally transparent, since you
2 ended up treating them differently in the report, I
3 would suggest you have 36 things in --

4 DR. MARTY: Okay.

5 DR. GLANTZ: -- Table D. That way it's all --
6 and the other -- make sure all the tables kind of fit
7 together.

8 Then the other question I had is if you look at
9 the 35 or 36 compounds in Table D, you drop out 20 of
10 them. And I have two questions, one for you and one for
11 the panel. And, that is, does anybody disagree for the
12 panel, that is, did they drop anything out that you
13 think they shouldn't have dropped out?

14 And the question for Melanie is to just -- if
15 there's anything more worth saying about why you dropped
16 the 20 that you dropped to get from Table D to Table 1
17 in the report? So I think that -- I'd like to hear a
18 little bit about that process with a few specifics and
19 then see if the panel agrees.

20 DR. MARTY: What drove the choice of the 11 was
21 evidence for differential effects either in children
22 versus adults or in young, experimental animal versus
23 mature, experimental animals.

24 DR. GLANTZ: Okay. So the 11 that you picked
25 then were the 11 where you had strongest reason to think

1 that was the case?

2 DR. MARTY: Yes.

3 DR. BLANC: But think about it from my
4 position, sitting here with the -- your detailed
5 chemical substance-by-substance Appendix B of the 11
6 that you chose. How am I supposed to scientifically
7 review your decision of those 11 versus the other 25?
8 Because OEHHA said so?

9 I mean, from my point of view, just give me
10 some guidance here. How am I supposed to accept the
11 decision that manganese compounds, which made it into
12 the 36, were excluded from the possibility of being in
13 the 11. And that I agree with the rationale for that --
14 I mean, I don't think, you know, seeing 97, you know,
15 reiterated case summaries because actually you didn't
16 get detailed evaluations.

17 So you go through and you said, okay. You've
18 explained the rationale for how you got down to 35 for
19 which you then contracted out to have, you know, fairly
20 detailed evaluations. I don't know whether I agree with
21 what your -- what the --

22 If, for example, again to use manganese as an
23 example, if the point is that there have been good
24 animal studies looking at neonatal equivalent exposure
25 and deficits with manganese and they've been negative

1 studies or whether the issue was that you don't feel
2 there's been enough animal data to look at preferential
3 neurotoxicology and the developing nervous system of the
4 appropriate animal model, I mean that to me would have a
5 very -- those are two very different scenarios; right?

6 I realize that with -- particularly with the
7 organified manganese, the data are only emerging now and
8 are quite limited. But clearly it's a huge, huge, huge
9 public health issue. And I would want to know exactly
10 what the basis was for excluding it.

11 Similarly, carbon disulfide, very large air
12 emissions, very important neurotoxin, very important
13 vascular toxin, very important peripheral known toxin.
14 What is the basis for which that fell out? Is it
15 because you couldn't find an animal study in the
16 literature, or there are ten animal studies all of which
17 are negative for differential effect?

18 DR. MARTY: Primarily, it's because of the lack
19 of data to describe a differential effect.

20 DR. BLANC: And in those situations, did you
21 have a clear policy for when you would -- so you have
22 this policy that you've taken, which we haven't got to
23 yet, on asthma, and, ipso facto, the airways are
24 narrower; therefore, anything that is an irritant you
25 will assume has a preferential effect, and maybe you

1 have one sort of semi-study of secondary data analysis
2 of, you know, a cohort from Arizona that suggests that
3 kids have peak flow in environments where one of the
4 things that was measured was formaldehyde.

5 But you've got this -- it's very heavily driven
6 by the assumption which you're about to get to in the
7 following slides about asthma. But I could make
8 certainly the same assumptions about anything which is a
9 neurotoxin that affects, preferentially, areas of the
10 nervous system even if I don't have great animal data
11 showing that pups are going to do worse than, you know,
12 six-month-old animals.

13 DR. MARTY: Well, maybe I should flip the
14 question back. If you have strong evidence, you have
15 the studies that show in pups neurotoxicity for chemical
16 X but for neurotoxin Y you don't have that information,
17 to us, the fact that you had specific studies was a
18 stronger indication of a differential effect than the
19 general assumption, which lots of people make that
20 neurotoxins are going to be worse in young animals.

21 DR. BLANC: But your review of your substances
22 made it by being neurotoxins. In fact, the only one in
23 the top five is lead and mercury on the bottom. Those
24 are the --

25 DR. MARTY: Okay. Then what the difference --

1 what we have to factor in is exposure. So where we had
2 strong evidence of exposure, then that also propelled
3 something higher up in the chain.

4 For mercury, there's lots of exposure, but it's
5 mostly from fish or water borne pathways in California.
6 There are some hot spots of exposure in terms of
7 airborne.

8 DR. BLANC: Okay. But I'm not arguing about
9 mercury. Mercury made it into the 11. I'm talking
10 about the things that didn't even make it into the 11.
11 They're not even on the radar screen anymore.

12 DR. MARTY: That was primarily a lack of direct
13 studies looking at --

14 DR. BLANC: Rather than studies that were done
15 that were negative?

16 DR. MARTY: Yeah.

17 DR. BLANC: And that's a big difference, isn't
18 it, from a public health point of view? So these are
19 chemicals which are presumed innocent until proven
20 guilty.

21 DR. MARTY: Yes. But it comes back to we have
22 to pick five.

23 DR. BLANC: I understand you have to pick five,
24 but first --

25 DR. MARTY: So which five are we going to pick?

1 The ones that are presumed innocent until guilty?

2 DR. BLANC: Well, I don't know. I can sit here
3 and make the argument that I think neurotoxicity would
4 drive things a hundred times more than the issue of
5 whether an irritant would cause airways to be narrower
6 in children. And I could also make the argument, and I
7 just may be a little out of order, but I know that you
8 weighted things towards developmental -- prenatal
9 developmental effects drove some of these things.

10 DR. MARTY: Actually, most of it's postnatal.
11 Some prenatal.

12 DR. BLANC: Some prenatal.

13 DR. MARTY: You're right. You know, from a
14 scientist, you have to worry about both in utero and
15 postnatal. But I understand your point, and you can see
16 how hard it was for us to do this.

17 DR. BLANC: I understand. But I can't see --
18 because we're talking about it, but I can't see from the
19 document.

20 DR. GLANTZ: I think the question I would ask
21 to you is you prepared these 35 reviews -- or I would
22 say 36 since you split the PCBs into two groups. I
23 mean, is there any reason that you couldn't in the next
24 iteration of this document include those or have an
25 appendix document or something so people can see what

1 there is to see? You know?

2 And then I think -- I think that if that
3 wouldn't be like a horrible, onerous thing to do, I
4 think that would help. And then I think to just have a
5 little -- what I would do is I would take Table D and,
6 you know, break it into two parts.

7 And, you know, it's just -- you know how we're
8 sort of winnowing to make it very explicit? I'd like to
9 see a table with the 25 that aren't in the 11 with just,
10 you know, if it's in Table 1, you have, like, endpoint
11 of most concern and major reasons why chosen, which I
12 think is very helpful, and I think it would be useful
13 for the 25 to just have a table and say why didn't this
14 make it into the top 11? Just a sentence or two.

15 CHAIRMAN FROINES: Well, I think it could be
16 even easier than that in some ways. I think that part
17 of the problem comes -- I bet you when you contracted
18 these out, you got these literature reviews back, and
19 they weren't sufficiently focused on the issues. You
20 had broad -- you got broad reviews back when, in fact,
21 what we're asking is a very precise question. Is there
22 evidence for differential susceptibility? That's the
23 question.

24 And so the question -- you could do it with --
25 almost with a table, which is, is there evidence for

1 differential susceptibility for the 35? Yes or no. Is
2 there -- is there evidence lacking? Yes or no. Which
3 goes to the question of chemicals are innocent until
4 proven guilty. What is -- what are five references
5 where the answer to one of those -- the first question
6 is yes, what are the five references that would document
7 that answer? And that's it. You've done it.

8 DR. GLANTZ: Yeah. That would be fine, too.

9 CHAIRMAN FROINES: It actually is a very
10 straightforward task, if you have a focused agenda. If
11 you want to review the toxicity of arsenic and it's --
12 and you get a document that's full of all this stuff
13 that has no relevance whatsoever to the question at
14 hand, then, in fact, it's going to become more difficult
15 to wade through.

16 So my sense is that the question about the 35
17 is not so difficult if a very focused criteria is
18 established and then answered accordingly with
19 references and with primary references, not secondary
20 reference.

21 DR. GLANTZ: Yeah. I mean, that would be
22 acceptable to me, too. But, again, I think you just
23 want to make it very -- and, see, then that way people
24 could look at it and say, "Okay. I understand why you
25 drew this conclusion and why you narrowed it down to the

1 11." I mean, I agree with you.

2 Ultimately, you're getting to five, but I think
3 at each step of the way, the rationale needs to be very
4 clear. I mean, I don't think anybody here today has
5 said that any -- that the basic approach you've taken is
6 not really reasonable. I think it's quite reasonable.

7 But for the document to stand, all of this
8 needs to be spelled out in sufficient detail for people
9 to just understand exactly, you know, what you did. And
10 if people want to come in and argue, then they can argue
11 about specific issues, you know, rather than feeling
12 like they're shooting in the dark.

13 CHAIRMAN FROINES: I can give you a very good
14 example of this I think, and, that is -- I think Paul
15 would agree -- that it's not clear to me why hexane is
16 not on the list. Hexane is a compound with very high
17 exposures, and it's certainly a powerful neurotoxin.
18 And, as Paul said, one can make an argument as
19 neurotoxicity as being equally an important defining
20 feature as asthma is, given the developmental issues in
21 post-utero periods of time or in utero.

22 So that it's not -- it's not obvious to me why
23 formaldehyde is on the list and hexane isn't. It's not
24 exposure. It's not the level of evidence. So somebody
25 made a decision that is clearly not transparent.

1 DR. BLANC: Well, let me give you another
2 example. So that, I mean -- something that I can't see
3 here, so I need to see where it fell out. Let's take
4 something that's in the 35, which is methylene chloride.
5 Methylene chloride is metabolized to carbon monoxide.
6 It's one of its main toxicity issues.

7 Neonatals have -- neonates have a higher
8 concentration of fetal hemoglobin, which binds carbon
9 monoxide much more avidly than other kinds of
10 hemoglobin, which is why in-utero exposure to carbon
11 monoxide, for example, is more of a problem for the
12 fetus than for the mother.

13 Wouldn't -- and there's a fair amount of
14 sources of exposure to methylene chloride, so there's
15 something where you have clear -- now, you may not --
16 you're out source reviewer may not have found a study
17 with neonatal pups exposed to methylene chloride, but I
18 don't need that study because I already that it's
19 metabolized to carbon monoxide, and I know from other
20 studies that carbon monoxide differentially affects
21 neonates.

22 Is that a level of review that happened
23 secondarily in OEHHA that you're confident that things
24 didn't fall through the cracks?

25 DR. MARTY: We did take those types of

1 considerations into account. But say for your example
2 of methylene chloride, the exposures in ambient aren't
3 going to produce much carbon monoxide.

4 DR. BLANC: No. But you're supposed to take
5 into account criteria air pollutants plus exposure to
6 these things.

7 DR. MARTY: Right.

8 DR. BLANC: So it wouldn't take much methylene
9 chloride, would it, added to the ambient levels of
10 carbon monoxide potentially?

11 DR. MARTY: Well, you'd have to do a kinetic
12 analysis, knowing exposures and the rate of carbon
13 monoxide formation and how much that adds to the carboxy
14 hemoglobin load.

15 DR. BLANC: And I'd have to see your appendix
16 where you said that we did that and --

17 DR. MARTY: We didn't do that. How could we do
18 that in the time that we had?

19 DR. BLANC: Well, then maybe it should be 12
20 because you say we have good reason to suspect that it
21 should make it onto the radar screen. Or maybe there
22 should be 36, and you should never have tried to do the
23 Tier 2.

24 You should just -- I mean if you can't sit here
25 and tell me that you have such a lack of data and yet

1 scientific rationale for A, B, C and D but you know --
2 you know, it goes back to the old saga of I dropped my
3 keys over there but I'm looking over here because this
4 is where the light is on.

5 DR. MARTY: Okay. Let's back up for a second.
6 Just comment one, Tier 2 does not mean that's the next
7 five in line. That's not what that means. Okay? It
8 means that those rose to the top based primarily on
9 toxicity information. We were concerned about them, but
10 they didn't make the top five. Now, some switching can
11 go on because there was good reason that they actually
12 got to the top 11.

13 In terms of the rest of the chemicals that
14 didn't make it on, we really -- for this go around, for
15 the first set of listing, wanted strong, toxicology data
16 or epidemiology data to get them on the list. We're
17 going to be looking at all of the TACs under this
18 statute. So the list will be updated over time, but we
19 felt compelled for the first go around to really have
20 strong information.

21 We can make cases for a lot of chemicals based
22 on just the kind of analogy that you just did for
23 methylene chloride. But where you compare methylene
24 chloride to lead, the weight of the evidence for lead is
25 huge.

1 DR. BLANC: I'm comparing methylene chloride to
2 formaldehyde, quite frankly.

3 DR. MARTY: Okay. Well, even if you compare --
4 at least for formaldehyde, we actually had studies that
5 looked in kids.

6 DR. BLANC: You had one study of peak flow in
7 kids and a community study where it was one of a variety
8 of things. You know variety and chamber studies of
9 formaldehyde which don't particularly suggest that
10 asthmatics are more sensitive to formaldehyde than
11 anybody else, so --

12 DR. MARTY: We do have evidence that
13 formaldehyde at low levels impacts lung function in
14 kids. In only one study did they compare adults and
15 children, and in that study, the authors concluded,
16 based on their data, that the adults in the same
17 households were less affected. It's a complicated
18 study. There's no doubt about it. But there we
19 actually we had a piece of information --

20 CHAIRMAN FROINES: It's not a complicated
21 study. It's actually a simple study.

22 DR. MARTY: I should not have said that. But
23 my point is that we had information there for
24 formaldehyde. I don't have an equivalent set of studies
25 for methylene chloride. This is not to say we're never

1 going to look at methylene chloride.

2 DR. BLANC: But you don't need --

3 DR. MARTY: It's --

4 DR. BLANC: But you don't need the same studies

5 because the biological issues are so different. I mean,

6 I'm not harping on methylene chloride per se, but I'm

7 trying to use it as one example. There are so few

8 examples where there is absolutely clear cut biological

9 reasons why an infant would have more toxicologic

10 susceptibility than an adult aside from all of these

11 sort of very generic issues that we're dealing with.

12 DR. MARTY: It's -- you know, I can't not agree

13 with you. This is a real struggle because you can build

14 cases -- similar cases for other chemicals.

15 CHAIRMAN FROINES: Part of the question is how

16 does -- for example, we just gave two examples which I

17 think are reasonable, hexane and methylene chloride.

18 How do they end up not on the Tier 2, and non-planar

19 PCBs do occur, and there's -- and the level of exposure

20 of non-planar PCBs at this point in history is

21 vanishingly small.

22 So here you have hexane, which is in gasoline

23 and a whole bunch of other things, and so you have

24 relatively significant concentrations, the atmospheric

25 chemistry notwithstanding, and you clearly have evidence

1 of powerful neurotoxicity. How does a non-planar PCB
2 get on this list and hexane doesn't? I don't get it.

3 DR. MARTY: Again, it would be based on studies
4 in the literature that looked at impacts in either young
5 animals or children. In the case of PCBs, it's both,
6 young animals and children. But we don't have those
7 equivalent studies for hexane at least that popped up
8 during the focus literature review. I don't feel
9 that --

10 DR. GLANTZ: I guess -- go ahead.

11 DR. BYUS: I have one question that harkens
12 back to the generic differences between children and
13 adults. How much of that has taken into consideration
14 the uncertainty factors when we do the original
15 calculations, say, for the RELs and the cancer potency?
16 I mean, aren't the uncertainty factors supposed to
17 consider those differences, and then how does that fit
18 in?

19 DR. MARTY: That's the reason, yes, that we
20 used that.

21 DR. BYUS: But it should say that in here.
22 It's like we're not ignoring all those things when we do
23 risk assessments for the differences between children
24 and adults. The uncertainty factors are supposed to
25 take that -- some of these things into consideration.

1 Am I wrong?

2 DR. MARTY: No. That's correct. Particularly,
3 the tenfold inter-individual variability factor.

4 DR. BYUS: Right.

5 DR. MARTY: We have actually a whole other
6 project going to look at whether that tenfold is
7 adequate for some sets of chemicals. But you're right,
8 and I don't think we mentioned that.

9 DR. BYUS: You didn't.

10 CHAIRMAN FROINES: Stan.

11 DR. BYUS: You should mention that because it's
12 important because even though you might not have used
13 these differences between children and adults in
14 construction of this list, those things are, in fact,
15 considered when you do the normal risk assessments with
16 the uncertainty factors.

17 DR. ALEXEEFF: Let me just --

18 CHAIRMAN FROINES: Stan.

19 DR. GLANTZ: I'll wait one second.

20 DR. ALEXEEFF: I just have a comment in regards
21 to Dr. Blanc's comment. As Melanie indicated, we're not
22 going to ignore the rest of the substances on this list,
23 the rest of the TACs. This is sort of step one of the
24 process. We're expecting that in a couple years to
25 basically come back with all the other ones evaluated.

1 But before we can do that, before we can do
2 that, we have to develop the criteria. You know, what
3 are the issues? For example, the metabolism issues and
4 stuff like that. We tried in the beginning here to pick
5 the more straightforward ones of which there were data.

6 In fact, when this whole bill was being
7 discussed, we were reticent to preparing any list prior
8 to developing all of the criteria, but the law was
9 passed with the requirement for a list before we could
10 actually develop all the reasons for why something
11 should be on the list. I mean, it just takes time to
12 lay it out and come up with all the different
13 mechanisms.

14 So we tried to pick those that we thought were
15 the most straightforward, and so I think part of this
16 dialogue is helpful because it will tell us which types
17 of mechanisms we need to go back and look at to lay out
18 and develop the guidelines or come back with the revised
19 list, not in the next couple months or month, whatever
20 the time line is, but I'm talking about the -- in the
21 year's time frame. That's actually in the statute as
22 well.

23 But -- so I think hopefully the table that we
24 prepare will clarify some of these issues as to why it
25 didn't make it to the top 11. And that's to say it

1 wouldn't be something we can't disagree about or have
2 different opinions, but at least it will be clear as to
3 why it didn't make it, and hopefully we can clarify
4 that.

5 CHAIRMAN FROINES: I think it's important to
6 say that we understand that you operate under a tight
7 time frame and were basically doing the best you could
8 under the circumstances given that we have a July 1st
9 deadline for the first five. So I don't think anybody
10 at this table is not appreciative of the short time
11 frame that you're operating in and the level of effort
12 that's required.

13 And the tension comes because this is such an
14 important process that everybody's trying to do it
15 right. And clearly it's going to get much better as we
16 go down the road when you have a time to develop these
17 documents in a more thorough and careful way.

18 And so what's happening is that people are
19 critical of the -- of what was produced. I think that
20 goes without saying. And -- but it's intended to set
21 the process right so that we have everything as clearly
22 defined as possible as we move down the road so that
23 this panel can do its job adequately and that people who
24 represent the public and various interests can
25 understand what's going on.

1 So I think the context is a supportive one, but
2 it's also a critical one, and we're going to be very
3 critical for the rest of the day. And -- but, again,
4 it's within that context, so nobody needs to feel as
5 though we don't understand that this wasn't a difficult
6 exercise.

7 But I do think that it's really important that
8 we do better on defining criteria and the basis for
9 decision making, which is what's been said a number of
10 times.

11 DR. GLANTZ: I'd like to come back to the list.
12 I understand what you're saying, George and Melanie, you
13 know, that Tier 2 doesn't necessarily mean that that's
14 the next five. But as a practical matter, I think if
15 you read the public comments and the people in the
16 audience, you know, the people who make acrolein would
17 rather not see it on the list at all, you know -- or I
18 just picked that out because it was the top one.

19 And I think the question -- I mean, I have some
20 concerns about which is in Tier 1 and Tier 2 of those
21 11, but I think before we get to that, I think it would
22 be worth asking the panel: Is there anything that's in
23 Tier 1 or Tier -- and we don't -- it doesn't have to be
24 11, but I'd rather it wasn't 35, you know? I think we
25 want to table the report to be the ones that are deemed

1 the most important, and, you know, maybe some new
2 information will become available over the next year or
3 two that will make you want to change that.

4 But I think the question is: Is there anything
5 people think is in Table 1 in the report, the ones that
6 they've picked as the top 11, that doesn't belong there
7 in the top 11 or 12 or whatever we thought was
8 reasonable? And is there anything that's been in
9 Table D that isn't in the report that ought to be,
10 without throwing the whole list in?

11 I think, you know, the prioritization process
12 is an important one, so I think trying to keep these
13 lists about how long they are is a good idea. But
14 several things have been kicked around. They're not
15 things that I personally know a lot about, so I just ask
16 the panel: Is there anything on Table D that isn't in
17 Table 1 in the report that we think ought to be looked
18 at? That it ought to be.

19 And, conversely, is there anything -- I've
20 heard some comments about formaldehyde, for example, and
21 seem to suggest maybe it shouldn't be given a priority.
22 So I'd be interested in any comments. You people know
23 more than I do.

24 CHAIRMAN FROINES: I think Paul's point,
25 though, is well taken insofar as that question, in a

1 sense, presupposes that we've had a chance to look at
2 some of the reviews in the literature.

3 DR. GLANTZ: Yeah. But there's going to be --
4 there's going to be another draft of this. But I think
5 in order to give OEHHA some guidance, is there anything
6 that people think, you know, ought to really be
7 seriously --

8 CHAIRMAN FROINES: To answer your own question,
9 do you?

10 DR. GLANTZ: I don't. I would move some things
11 around on the list of the report. I don't. I mean,
12 does anybody else?

13 CHAIRMAN FROINES: Craig?

14 DR. BYUS: Uh-uh.

15 CHAIRMAN FROINES: Roger?

16 DR. BLANC: Well, I do. And I'm going to put
17 it in a slightly toned down version. I have things for
18 which I would be so concerned that it would be -- I
19 would be going through a -- an appendix thing, and I
20 would be getting on Medline and making sure that
21 something hadn't been missed. So let me tell you each
22 of those and why, and some of them I've already
23 mentioned.

24 I would be extremely concerned about carbon
25 disulfide because of its cardiovascular effects and

1 because of its central nervous system toxicity and
2 because I know that it's out there as an important
3 ambient.

4 I would be concerned about chlorine because
5 it's -- other than sulfa dioxide, it's the only other
6 chemical for which there's good evidence that person's
7 with airway hyperactivity have a more extreme response,
8 and; therefore, if there's any chemical on the list for
9 which asthma ipso facto is going to be something that
10 you're going to then say "Children must be doing worse,"
11 chlorine would be one of the chemicals. And, in fact,
12 the REL for chlorine is based on the response of people
13 with airway hyper-responsiveness.

14 I would be extremely concerned about manganese,
15 even if levels in the atmosphere currently are trace
16 because we have an extremely important reason to --

17 DR. BYUS: Paul, go back to the last thing you
18 said about chlorine. If that's what the REL is based
19 on, then would you need the extra considerations for it?

20 DR. BLANC: According to this, yes. I mean, as
21 I read the statute --

22 DR. BYUS: Okay.

23 DR. BLANC: -- it doesn't even matter.

24 DR. BYUS: I'm sorry.

25 DR. BLANC: No, no. Methylene chloride for the

1 reasons that I've said in terms of carboxy hemoglobin
2 and in terms of the statute's requirement to consider
3 interactions for priority air pollutants.

4 And those are the ones I believe that I would
5 want to look at more closely.

6 DR. GLANTZ: Is there anything on -- in Table 1
7 in the report that you think probably shouldn't be there
8 compared to these other things?

9 DR. BLANC: I actually don't want to -- I'm not
10 prepared at this point to discuss it from that.

11 DR. GLANTZ: Okay.

12 DR. BLANC: Because I think the -- I'm
13 taking -- we have enough data to review whether or not
14 they felt that there was enough to rise up, at least
15 into some group that needs to be considered as a
16 candidate for the five.

17 I'm really addressing a much different question
18 which is -- and I take what you say at face value that
19 because something isn't among the 11, it doesn't mean
20 that it won't get looked at closely, but let's be
21 realistic. It's going to be a harder sell a year from
22 now to then suddenly move something up from being off
23 the radar screen to being something -- I mean, I'm just
24 looking at it from sort of a public health point of
25 view. So I think this is not a trivial question

1 necessarily.

2 DR. ATKINSON: But then that does point out the
3 need to -- I think you need to put some more verbiage
4 about the ambient concentration data. I mean, you just
5 say that you take it from ARB's database. I think a
6 little more discussion of what that database includes,
7 the air basins it was taken in and so on and some
8 caveats that, you know, it's not -- may not be -- may
9 not really be correct, and there may be interferences,
10 and there may be and are data from other studies which
11 may really supersede those.

12 DR. BLANC: I guess one technical question,
13 Melanie. Methyl bromide, which is No. 23, but is -- it
14 is a fumigant, but it has other uses, and that's why it
15 was allowed to stay here because everything else --

16 DR. ATKINSON: It's a pesticide.

17 DR. BLANC: Everything else has fallen off.

18 DR. ALEXEEFF: That's right. Methyl bromide is
19 on the list because there is the Hot Spots Law which
20 requires permitting of stationary sources of which
21 fumigations chambers are stationary sources.

22 DR. BLANC: So ARB does --

23 DR. ALEXEEFF: That's why that one --

24 DR. BLANC: So ARB does --

25 DR. ALEXEEFF: Well, the air districts do that,

1 yeah. So that's why that is on that list.

2 DR. BLANC: Well, then that's probably another
3 one I would say I would be very suspicious about of the
4 list.

5 And, John, maybe you have some others,
6 particularly some of the other heavy metals that I
7 haven't talked about.

8 CHAIRMAN FROINES: Gary?

9 DR. FRIEDMAN: No, I can't add anything.

10 CHAIRMAN FROINES: I want to avoid getting into
11 giving you a long list because I think it would be
12 useful to give you a long -- to expand the list of 11 to
13 a larger number, perhaps not 35, but a larger number,
14 but I don't -- I don't know.

15 Acetaldehyde fits into your generic issue of
16 small airways irritants, so it's obviously one for which
17 it could be on the list. In that sense, if you have an
18 expanded list, it probably should be on the list. But
19 then you have a problem with glutaraldehyde, for
20 example, and croton aldehyde. The aldehydes, given the
21 criteria of small airways and irritant effects, as we
22 know, there's a whole list of aldehydes that would fit
23 that criteria. But acetaldehyde would be one.

24 And then obviously the metals, arsenic, cadmium
25 and chromium are a second group of three, and obviously

1 I even pointed out hexane. But I'd rather sort of not
2 give you that as a list. I'd rather give you that as a
3 list based on looking to see if there's evidence of
4 differential effects. Do you see what I'm saying?

5 In other words, I think that I would expand the
6 list. Paul was actually making some decisions, for
7 example, with methylene chloride that he says that there
8 is evidence of a differential effect. And so,
9 therefore, that could reasonably be on a list without
10 necessarily going through all the toxico-kinetics and
11 metabolism issues that one might have to look at.

12 So I can give -- I can mention those compounds,
13 but I would rather look at the reviews and see to what
14 degree you think butadiene, for example, which is a very
15 important compound, has any evidence of differential
16 toxicity. And if it does, then I would put it on the
17 list. Do you see what I'm saying?

18 DR. ALEXEEFF: Well, I think --

19 DR. BLANC: Assuming that they did that and
20 there wasn't, I guess?

21 CHAIRMAN FROINES: I don't know that. No.
22 Because I don't agree with that. You assumed that and
23 there wasn't, but that's wrong when you consider hexane
24 because hexane there is evidence of differential effects
25 to the degree that you think of neurotoxicity as having

1 some generic elements to it.

2 DR. MARTY: I think just a couple comments. I
3 am not trying to be argumentative.

4 CHAIRMAN FROINES: That's okay. I understand
5 that all the generation may not necessarily have a
6 developmental characteristic. And so the mechanism of
7 CPDA does not necessarily give you evidence for a
8 developmental effect. I understand that with hexane,
9 but it's still -- the neurotoxicity question is still one
10 that needs to be evaluated.

11 DR. MARTY: All right. I agree. We debated
12 endlessly whether, for example, all neurotoxins should
13 be on the list because there's lots of reasons to think
14 a developing organism would be more sensitive to them.

15 Data -- genotoxic carcinogens, there's a lot of
16 mitosis going on. You would anticipate a larger number
17 of targets for mutation so forth and so on. And we did
18 have a lot of concern about chlorine, but when we looked
19 at chlorine concentrations in the air compared to the
20 reference exposure level against acrolein concentrations
21 in the air compared to the reference exposure level,
22 acrolein wins out.

23 DR. BLANC: I'm not making argument for
24 acrolein not to be on the list, am I?

25 DR. MARTY: No.

1 CHAIRMAN FROINES: We're also not clear why
2 your list has 11 chemicals on it. That's the question
3 that's being raised.

4 DR. ALEXEEFF: I think there's --

5 DR. BLANC: Before you answer that, George, I
6 just want to say that what is very confusing about your
7 last statement, Melanie, is that the way you explained
8 it is everything made it to D already because of
9 importance in its ambient levels to REL or its inherent
10 toxicity. And then the thing that makes something jump
11 from List D to the final 11 is levels of evidence of a
12 differential effect in kids.

13 And your answer about chlorine was, yes, there
14 is evidence that it would differentially affect
15 asthmatics and, therefore, the kids in our rationale,
16 but the concentration levels weren't that high in the
17 air. But --

18 DR. MARTY: It was also extent of exposure.
19 Within the list of 35 or 36, Stan, we also had concerns
20 about extent of exposure.

21 DR. BLANC: That would have kept something from
22 getting to the top 11, even though that's not what you
23 said previously?

24 DR. MARTY: Actually, it's on the slide.

25 CHAIRMAN FROINES: Yeah. But that doesn't -- I

1 mean, but how do you get with non-coplanar PCBs?

2 DR. MARTY: That's weighting heavily the
3 toxicity. Also PCBs are virtually everywhere in every
4 body.

5 CHAIRMAN FROINES: That doesn't mean a thing.
6 And in terms of -- I mean the notion of is there a
7 potential for exposure that somebody can then go do
8 something about as a public health issue is what this is
9 all about. This isn't about making decisions strictly
10 on the basis of toxicology. The idea is to protect
11 children and because -- and the way you protect children
12 is through various control mechanisms.

13 So if you have something that can be
14 controlled, then that's a consideration that goes into
15 the risk management phase, and I understand all that
16 rhetoric. But I still think it's the underlying
17 consideration. The underlying consideration of the law
18 is to protect children.

19 Therefore, if you have something that for which
20 the exposure may be very widespread but doesn't occur
21 through an ambient or airborne pathway, because we're
22 focused on air issues now, and resulting in a
23 contamination of soil, water or what have you, then we
24 have to be careful to put that as a high priority it
25 seems to me because it's not clear we can do anything

1 about it.

2 DR. MARTY: That's actually why it ended up in
3 Tier 2 instead of Tier 1. And Tier 2 just means those
4 11 that didn't end up in Tier 1, there's no other
5 significance to Tier 2. I agree with you. You know,
6 there was a case where we had strong epi and tox data,
7 but we felt strong enough to say, "There's differential
8 impacts here," but then when you go to look at the
9 exposure piece, you know there's exposure, but is air an
10 issue?

11 We think it's an issue for dioxins, but it may
12 not be the -- certainly it's not the driving pathway by
13 which you're exposed to PCBs.

14 DR. ALEXEEFF: I just wanted --

15 DR. GLANTZ: There's nothing, you know -- I
16 just want to reiterate, while the law says you have to
17 pick five, the law doesn't say you have to pick 11 for
18 this -- you know, and have your Tier 2. So it may be
19 that you might want Tier 2 instead of having six things
20 in it to have eight or nine.

21 I don't think -- I personally think because
22 of -- for the reason somebody made that once this is
23 done, it's going to be hard for things to jump into that
24 list. I mean, I don't think you want to put all 35 or
25 36 of these things in. I think you've gone through a

1 fairly rational winnowing process, but I think Paul
2 mentioned three or four more that ought to be seriously
3 looked at, and it may be that in the final report
4 instead of 11, there's 15, you know? Plus what
5 everybody else says.

6 DR. ALEXEEFF: There's -- you know, in terms of
7 reaching the group of 35, a lot of the general type of
8 issues were on the minds of the staff in terms of
9 putting them there, such as the issue of methylene
10 chloride in terms of metabolism in carbon monoxide.
11 That actually was certainly discussed. And the
12 manganese and a lot of those chemicals were put in the
13 top 35 because of knowledge of the general type of
14 issues.

15 And then -- and so I think once we put that on
16 the table, that could help clarify as to why it made the
17 top 35.

18 DR. GLANTZ: Yeah.

19 DR. ALEXEEFF: And then the next question is,
20 well, how come it made -- it did or did not make it to
21 the 11? And I think that what we could do for the ones
22 that you -- we've counted seven, six or seven. We could
23 provide additional summaries. Seven compounds have been
24 mentioned here.

25 CHAIRMAN FROINES: More if you take mine.

1 DR. ALEXEEFF: I wasn't taking -- you said you
2 weren't really proposing all of yours, but let me just
3 finish my sentence.

4 CHAIRMAN FROINES: But it's a rhetorical
5 statement; right? It says "arsenic." I want to know
6 whether or not your summary has evidence for
7 differential effects, and if there's evidence, then put
8 it on.

9 DR. ALEXEEFF: Yeah. We'll put that in the --
10 you'll see it in the generic type of table; okay? And
11 explain why it drops out. But we could do is provide
12 summaries for a number of additional ones for which
13 there is some evidence on that. The question comes with
14 something like if -- and I can't remember methylene
15 chloride, but if it's more of a mechanistic inference,
16 but there isn't really actually any studies we can come
17 up with -- although, it's, you know --

18 DR. BLANC: Well, I would say that that would
19 be an example for something where the logic is so
20 concrete that you don't actually need the specific
21 study.

22 DR. ALEXEEFF: Yeah. Well, that's kind of the
23 question.

24 DR. BLANC: If A equals B and B equals C then A
25 equals C. So if you have studies that show that it is

1 metabolized to carbon monoxide, which you do, and if you
2 have other studies of carbon monoxide which show that
3 there's a differential effect in children, which you do,
4 then I don't think you need the study of methylene
5 chloride in children.

6 DR. ALEXEEFF: Right. Anyway, we can prepare
7 summaries for a number of those compounds and then the
8 panel can decide whether they're relative to --

9 CHAIRMAN FROINES: When you add in carbon
10 monoxide.

11 DR. BLANC: Yeah. And there has to be some
12 comment on -- you know, some REL-type argument about,
13 you know, potential for exposure to carbon monoxide as a
14 particulate air pollutant.

15 DR. ALEXEEFF: We've done carbon monoxide under
16 the other part of this particular statute. We just
17 completed a complete review of carbon monoxide.

18 DR. BLANC: And then, for example, for
19 manganese, which of all of these I guess I would make
20 the argument that is the one where you have the most
21 chance to have a real public health impact from this
22 document. And if the whole reason why -- I mean, I want
23 to look very closely that you have absolutely no -- you
24 know, data, other than inferential data, of any
25 susceptibility of young animals to manganese. Because

1 if what kept manganese off this list does not have
2 enough current air pollution data for manganese, then I
3 would say -- I put it No. 1 of the 5 because that's the
4 one you don't want to have the air pollution for.

5 DR. ALEXEEFF: Actually, there is ambient data
6 on manganese. It's on the table.

7 DR. GLANTZ: But the point Paul is making is
8 he's concerned that there's going to be a lot more.

9 CHAIRMAN FROINES: It's an additive issue.

10 DR. MARTY: You know what? I thought we had --
11 Kirk, is there not a statute banning the use of the
12 organo-manganese compounds in gasoline in California?

13 MR. COLLINS: Yes. In 1977. We've got copies
14 of it because this came up a couple years ago. There is
15 a statute banning the use of it.

16 DR. BLANC: The EPA hasn't banned it yet.

17 MR. COLLINS: Correct.

18 DR. MARTY: That's right.

19 DR. ALEXEEFF: But California has.

20 DR. MARTY: And that -- you know, we have the
21 same, identical concerns about manganese. That compound
22 makes me nervous, and I think it's nuts, personally, to
23 put it in gasoline.

24 DR. ALEXEEFF: But what we could do is we could
25 research that issue, and if that's our reason for not

1 putting it on the list, that it's not going to be used
2 in gasoline, we could lay that out, you know, the
3 statute, cite that, and we can clarify that issue.

4 DR. BLANC: And were that statute ever to be
5 reversed it would --

6 DR. ALEXEEFF: That would be what the
7 information is.

8 DR. FUCALORO: You're talking about -- excuse
9 me. I'm sorry I'm late.

10 DR. BLANC: We're talking about this list --

11 DR. FUCALORO: Manganese.

12 DR. BLANC: -- here and why things that are on
13 this list aren't among the -- that only 11 of these --

14 DR. GLANTZ: Does Gary have anything to add?

15 DR. FRIEDMAN: No, I have no other comment.

16 CHAIRMAN FROINES: Melanie, tell me -- why
17 don't you -- I don't think we can get to your asthma
18 slides before lunch, so why don't you finish this phase.

19 DR. GLANTZ: Are we going to talk about Tier 1
20 versus Tier 2 before lunch?

21 CHAIRMAN FROINES: No. It's almost
22 1:00 o'clock.

23 DR. MARTY: I think we should go chemical by
24 chemical --

25 DR. GLANTZ: Okay.

1 DR. MARTY: -- to do that.

2 This slide is just what you already know, in
3 alphabetical order, the proposed listing, which is
4 equivalent to Tier 1 as described in the document. And
5 then, Andy, the next slide just shows those that fell
6 out and didn't make it to Tier 1. And then I have
7 asthma slides which I --

8 CHAIRMAN FROINES: We can come back to the
9 asthma slides, but I think Stan would like to talk about
10 Tier 1 versus Tier 2, but my assumption is that that
11 would be best done as we go through the level of
12 evidence on the individual compounds, but if he wants
13 to --

14 DR. GLANTZ: Whatever.

15 CHAIRMAN FROINES: -- argue it differently,
16 that's fine.

17 DR. GLANTZ: Just so we get to it. Whatever
18 you want.

19 CHAIRMAN FROINES: Well, it's whatever the
20 panel really wants.

21 DR. FRIEDMAN: I think it would be helpful to
22 just -- if it's not a long topic to deal with that
23 before lunch.

24 DR. GLANTZ: I think that diesel exhaust should
25 be in Tier 1. It's a fairly brief comment. The -- I

1 mean, when you read the report, you know, there's a lot
2 of, kind of, perseverating about why it isn't in Tier 1,
3 and I think that it belongs there. I think it's very
4 potent.

5 Several of the other things that are in Tier 1
6 are in diesel exhaust and I think that evidence it's
7 important and all the things we've been talking about
8 are very strong, and I think since we can only have
9 five, I would suggest that benzene be dropped down to
10 Tier 2.

11 Because I think in reading -- again, reading
12 the document and reading the public comments, I think
13 the -- that's -- of the things that you have there, some
14 of you guys know more about some of these other
15 compounds than I do or chemicals than I do, but that's
16 the one that I think if you had to pick one of those to
17 move down, that's the one I would suggest moving down.

18 CHAIRMAN FROINES: Elinor, you know, did her
19 thesis with Martin Smith at Berkeley, and she worked on
20 benzene, so she was strongly opposed to the benzene
21 thing.

22 DR. GLANTZ: Well, we normally have five. I'm
23 not saying we should all go out and drink benzene for
24 lunch.

25 CHAIRMAN FROINES: I was just joking.

1 DR. GLANTZ: Okay.

2 CHAIRMAN FROINES: I think you opened such a
3 pandora's box. That's why I would want to do it after
4 lunch when we actually have -- I meant it as data.

5 DR. FRIEDMAN: Yeah. I thought we were just
6 going to talk about criteria, not about specifics.
7 That's why I didn't understand.

8 DR. GLANTZ: Well, he asked me about the
9 specifics.

10 DR. FRIEDMAN: That's why I --

11 DR. BLANC: Well, we have a foretaste of what
12 we'll be discussing after lunch.

13 CHAIRMAN FROINES: Why don't we take lunch and
14 then have the asthma discussion. And then as one of the
15 criteria, which I think is what Melanie was planning as
16 an important criteria, and then go to the individual
17 chemicals. Does that make sense?

18 DR. GLANTZ: Where do we get lunch, and what
19 time do we need to be back?

20 CHAIRMAN FROINES: I don't know. Jim or Peter?

21 DR. BLANC: Downstairs in the cafeteria.

22 CHAIRMAN FROINES: Downstairs in the cafeteria.

23 DR. FRIEDMAN: John, may I request that we have
24 a rather short lunch because I have to leave at
25 3:00 o'clock, and I'd like to hear as much as possible.

1 CHAIRMAN FROINES: Half hour? Forty-five
2 minutes?

3 DR. BLANC: Thirty-five minutes.

4 DR. GLANTZ: We've got to find the place.

5 CHAIRMAN FROINES: Thirty-five. And we are
6 going to start at 1:35, so let's -- the panel should --
7 the audience doesn't necessarily have to do that, but
8 the panel members do.

9 (Luncheon recess.)

10 CHAIRMAN FROINES: Before we start,
11 Dr. Fucaloro would like to make a comment to the panel.

12 DR. FUCALORO: Yeah. I have to report an ex
13 parte contact with a gentleman who actually teaches a
14 course at our college in environmental law. He is an
15 environmental lawyer, and he's working for people who
16 reported in these contacts on the issue of lead.

17 So, basically, we discussed some of the issues
18 concerning inclusion on that list of five. And he, of
19 course, wants lead out. "Get the lead out," he told me.
20 But, of course, let the science do the talking. And we
21 just discussed it. I think his basic argument was what
22 was made in this report, which essentially states that
23 the toxicity of lead is high. Its -- the exposure level
24 is low. That's about it.

25 CHAIRMAN FROINES: Kirk, is there anything more

1 we need on this issue besides having it on the record?

2 MR. OLIVER: No.

3 CHAIRMAN FROINES: Well, that was the most

4 succinct exchange we've ever had.

5 DR. GLANTZ: Well, then I need to comment.

6 CHAIRMAN FROINES: This is where you would want

7 to fill it up.

8 DR. GLANTZ: Just joking.

9 Melanie is fortified and ready for another

10 round.

11 DR. MARTY: Are we ready?

12 CHAIRMAN FROINES: She's in danger of

13 developing pugillus encephalopathy by the end of the

14 day.

15 DR. GLANTZ: What is that?

16 DR. BLANC: Getting hit on the head too many

17 times.

18 DR. MARTY: It's the Mohammad Ali syndrome.

19 I'd like to start this afternoon by talking a

20 little bit about asthma because we use asthma --

21 exacerbation of asthma as a toxicological endpoint in

22 some of our arguments for differential susceptibility of

23 children versus adults. I just wanted to flesh that

24 argument out a little bit.

25 The asthma prevalence rates in children are

1 higher than adults. There's reasonably good statistics
2 on that from the asthma surveillance program at CDC. So
3 as on a population-wide basis, other things being equal,
4 if you exacerbate asthma in a population, you have more
5 kids likely being impacted than adults.

6 Also children have smaller airways. This came
7 up earlier in the discussion. So constriction of the
8 airway, which happens in asthma, will cause a greatly
9 increased resistance. The resistance is inversely
10 proportionate to the cube of the radius. So as you --
11 it's not a linear increase in resistance. It's quite a
12 bit more than linear.

13 So when you have a child with a small airway,
14 and they have an asthma attack in that the mucous
15 secretion blocks the airway as well as the broncho-
16 constriction, they can quickly get to the point where
17 the increase in resistance to air flow causes a very
18 severe problem in a child and less so in an adult who is
19 starting out with a larger airway.

20 I'd like to add also that hospitalization rate
21 data indicate that it's highest for the zero to four-
22 year-old age group, and I'll get to that in a minute.
23 And also asthma prevalence --

24 DR. GLANTZ: You mean hospitalization rates for
25 asthma?

1 DR. MARTY: Yes. Right.

2 DR. GLANTZ: Not hospitals --

3 DR. MARTY: It's actually based on discharge
4 data so -- and what the discharge data indicates what
5 the person is in the hospital for.

6 DR. FRIEDMAN: Are you saying that they're
7 highest among other -- taking zero to four year olds'
8 asthma rates are the highest cause of hospitalization,
9 or are you saying that given you have asthma, you're
10 more apt to be hospitalized if you're zero to four?

11 DR. MARTY: It's if you compare by age
12 groupings zero to four, five -- I forget what the age
13 groupings are. But the highest rates, according to the
14 discharge data are for zero to four year olds, and it
15 drops out as you get older.

16 DR. GLANTZ: But the question here is what's
17 the denominator?

18 DR. BLANC: Per 100,000 children.

19 DR. GLANTZ: No, no. Is this --

20 DR. MARTY: You know what? I have a slide on
21 that, so maybe we should talk about it when I get to the
22 slide.

23 DR. GLANTZ: Okay.

24 DR. MARTY: The asthma -- I just wanted to add
25 in that the asthma prevalence, at least in the U.S. and

1 elsewhere, is increasing. There have been large
2 increases over the last couple of decades. So it's an
3 important disease.

4 Next slide, Andy.

5 DR. FRIEDMAN: Wait. I have to -- can I
6 interrupt? I always want to acknowledge Stan's help in
7 asking my questions. Thank you, Stan.

8 DR. MARTY: Some of the discharge data, you can
9 get either on a national basis from the CDC, or the
10 Department of Health Services puts together a report,
11 asthma hospitalizations by county, and they break it out
12 by age and sex and race.

13 And if you look at overall hospitalization
14 rates, it's 216 per 100,000 discharges. So, in other
15 words -- right. The hospitalization rates for children
16 are much greater than for adults. The rates for kids
17 under one year old are three times that of the rates of
18 10 to 14 year olds, which goes hand in hand with the
19 smaller airway phenomenon.

20 Next, Andy.

21 DR. BLANC: Just say that the rate for
22 hospitalization is higher among children. I don't think
23 you can connect the dots and say it's because they have
24 smaller airways. Just -- you're certainly on firm
25 ground if you say that children -- per 100,000 children

1 have higher hospitalization rates in that age group than
2 in older age groups and leave aside the issue of the
3 airways with that.

4 DR. MARTY: Okay.

5 DR. GLANTZ: I still don't understand per
6 100,000. Is it per 100,000 children?

7 DR. MARTY: Discharges.

8 DR. BLANC: No, no. Per 100,000 children.

9 DR. MARTY: Mark is telling me it's per 100,000
10 kids.

11 DR. FUCALORO: In that age group, so that it's
12 standardized within the age group.

13 DR. MARTY: Yes.

14 DR. GLANTZ: So it's not per 100,000 hospital
15 discharges. It's per 100,000 --

16 DR. MARTY: No. It's per 100,000 kids.

17 DR. BLANC: For 100,000 children, the rate of
18 hospitalization is higher than per 100,000 adults.

19 DR. GLANTZ: Yeah.

20 DR. MARTY: And I did want to point out that
21 hospitalization, that's not doctor visits. That's not
22 going to the -- it's the doctor telling -- the doctor is
23 the person who puts you in the hospital. It's not you
24 going to the hospital saying, "I need to be
25 hospitalized." So I think it's a little more firmer

1 ground for looking at a differential effect between kids
2 and adults than just going to the doctor.

3 CHAIRMAN FROINES: I -- Melanie knows I'm going
4 to say this because she knows how I feel about it. I
5 think that the behavioral factors associated with going
6 to hospitals is so complicated that I think this is such
7 a poor example.

8 I mean, more -- you look at asthma rates, more
9 whites go to hospitals than blacks go to hospitals, but
10 that doesn't argue for a differential susceptibility of
11 whites over blacks. It has to do with socioeconomic
12 status.

13 DR. BLANC: I don't --

14 DR. MARTY: Can --

15 DR. BLANC: John, listen. I think you're
16 beating a dead horse. There's no -- no one doubts that
17 the rates of asthma among children, the prevalence. The
18 incidents of severity is higher among children than it
19 is among adults. Certainly until you get up to the very
20 old age of adults, and then it goes up again. So it
21 will be a question if you were talking about 75 year
22 olds.

23 But since your task is to say, you know, is
24 this a disease for which the rates are higher among
25 children, especially young children, that's not an

1 argument. That's not -- there are, of course, many
2 other diseases which are also at very high rates among
3 children compared to adults, but that's not the point of
4 this discussion either.

5 DR. MARTY: Right. Okay. Andy, next slide.

6 This is just a figure taken from this document of Age-
7 Adjusted Asthma Hospital Discharge Rates For Kids Ages 0
8 to 14 by Race and Sex. And, actually, African-American
9 kids have very high rates of hospitalization for asthma
10 relative to other race groupings. And that's discharges
11 per 100,000 kids and boys for some reason more than
12 girls.

13 DR. BLANC: And then it changes again but yes.

14 DR. MARTY: Next slide, Andy.

15 And this was just a different look at the data
16 by age group across a couple of years, and this is
17 California-specific data. Again, discharges per 100,000
18 by age. So the top line is less than 1, then 1 to 4,
19 then 5 to 9, then 10 to 14.

20 So this is the -- this is more data looking at
21 office visits per thousand by age group, for asthma. ER
22 visits per thousand by age group for asthma and
23 hospitalizations per 10,000 by age group. The groups
24 are big here: 0 to 4, 5 to 14, 15 to 34 and so on. So
25 I think that --

1 CHAIRMAN FROINES: I think I'm afraid I still
2 feel that Paul's right, that this is not an argument for
3 differential susceptibility to chemicals.

4 DR. FRIEDMAN: It is an argument for
5 differential impact, you know, on this group in terms of
6 the costs of their care and days lost from normal
7 activities. Is that part of the consideration?

8 DR. MARTY: Yes. Right. That's all we had
9 for -- in terms of introduction. We have a presentation
10 on each one of those 11 chemicals. We can start with --

11 CHAIRMAN FROINES: What's the -- what's the
12 conclusion -- having given that presentation on asthma,
13 what is the conclusion that you draw as a basis of your
14 criteria for defining susceptibility? What's the bottom
15 line from all that?

16 DR. MARTY: Well, the bottom line is that OEHHA
17 takes the position that things that exacerbate asthma
18 are going to have larger impacts in children on a
19 population-wide basis than on adults.

20 CHAIRMAN FROINES: Okay. Exacerbate asthma,
21 that's one criteria. That's different than evidence of
22 a chemical having irritant effect.

23 DR. MARTY: Yes.

24 DR. BLANC: Well, a generic irritant wouldn't
25 necessarily exacerbate asthma.

1 DR. MARTY: I think that the data show that
2 there are some irritants that don't necessarily
3 exacerbate asthma or that you can't see it in the
4 studies.

5 DR. BLANC: Well --

6 DR. MARTY: I don't think you could make that
7 argument that every irritant exacerbates asthma.

8 DR. BLANC: Are you -- well, let me ask the
9 question in a different way. Is what you're -- your
10 threshold then would be evidence that if you compared
11 asthmatics to non-asthmatics at the same exposure level,
12 that consistently the asthmatics would have a greater
13 increase in airway resistance in response to the same
14 concentration of the pollutant in question, and that's
15 what it would be?

16 DR. MARTY: That may be true, but that's not
17 our argument.

18 DR. BLANC: Then --

19 DR. MARTY: Our argument is that --

20 DR. BLANC: How would you then differentiate --
21 that's the way that I'm familiar with making the
22 argument that a chemical irritant -- because I think it
23 is a reasonable thing to say that most irritants would
24 tend to create a problem across the board in airways, if
25 they're water soluble particularly.

1 And, therefore, if you started from a narrow
2 caliber, the implications of having inflammation would
3 be worse if you already had narrowed airways. This
4 would be true generically for every single irritant and
5 would only be a matter of irritant potency.

6 If you're making the argument that, in fact, a
7 particular irritant would be more prone to induce
8 broncho-spasm in people with preexisting airway hyper
9 responsiveness, as opposed to people without preexisting
10 airway hyper responsiveness, then your list of
11 substances is vanishingly small. And, in fact, it
12 really is sulfa dioxide, sulfa dioxide and sulfa
13 dioxide, which is a criteria air pollutant.

14 If I had to then say beyond that what -- do I
15 believe that there is experimental evidence that is
16 consistently shown? And I would be very hard pressed.
17 There certainly has not been consistent evidence for
18 ozone, again, I grant you, as a criteria pollutant.
19 There has not been consistent evidence for nitrogen
20 dioxide, and it's been -- or oxides of oxygen, and it's
21 been a big area of controversy.

22 There certainly is not such experimental data
23 for formaldehyde or other aldehydes. And for chlorine
24 there's -- you know, there's one small study that
25 suggests that -- what a -- I know because I did the

1 study and am waiting for somebody else to repeat the
2 results. So that's why it's going to be --

3 DR. MARTY: That's why we're looking for
4 chemical-specific data.

5 DR. BLANC: -- difficult for me.

6 DR. MARTY: And it's in -- you know, when we
7 say something is exacerbating asthma, we're using
8 studies that show it exacerbated asthma rather than
9 saying "because it's an irritant, it probably
10 exacerbates asthma." That's where we're drawing a
11 distinction for this set of 12, 11 compounds.

12 DR. BLANC: And so, therefore, when we come
13 back to the individual studies, that would be the issue
14 that you are raising?

15 DR. MARTY: Right. Right. It's not that we
16 don't have concerns about some of the other irritants
17 for which there are direct studies, you know. It's not
18 that we're not worried about that. We are worried about
19 that.

20 DR. BLANC: But if you theoretically had an
21 epidemiologic study that had an association in a mixed
22 exposure and you had laboratory control human exposure
23 data that did not show the effect, wouldn't the
24 laboratory data argue more convincingly that the
25 epidemiologic association in the mixed exposure

1 situation was -- couldn't be used to single out,
2 perhaps, the substance that made you worry?

3 DR. MARTY: Well, it's a hypothetical. I'd
4 have to look at the studies. But I think it's also
5 important to remember that we're supposed to consider
6 multiple pollutant exposures. I mean, it's difficult to
7 say in a lot of the pollution epi studies which
8 pollutant is the worst actors. Probably interactions.

9 DR. BLANC: And then in terms of your rank,
10 hierarchy of conditions and -- for which particular
11 concern would be important among the pediatric
12 population, so hospitalization rates for upper
13 respiratory infection are probably higher among young
14 children than among adults, by and large.

15 DR. MARTY: I'm recollecting that that's the
16 case.

17 Mark?

18 DR. BLANC: So, therefore, if there was an
19 irritant pollutant that was associated with a greater
20 risk of upper respiratory, secondary infection, then
21 that would also be by the same logic something that
22 would be relevant?

23 DR. MARTY: Yes.

24 DR. BLANC: And if something, theoretically,
25 was associated with aggravated hyperglycemia, since

1 hospitalization rates -- were hospitalization rates were
2 to be higher, or juvenile diabetes, which it probably
3 isn't, but opposed to adult onset diabetes, since it's
4 such a big, burden disease. But I'm just saying it's
5 not specific.

6 There's nothing peculiar about asthma -- for
7 asthma per se. It's just that it's -- one, it's a
8 common disease, and it's a common disease among
9 children, and for children less than four,
10 hospitalization rates are higher. But were there to be
11 other conditions that were in the same category, they
12 would also logically be on the same level on concern.

13 There's nothing inherently about asthma that
14 has your attention in terms of --

15 DR. MARTY: It has our attention because we're
16 dealing with airborne pollutants, some of which we know
17 exacerbate asthma.

18 DR. BLANC: Well, no.

19 DR. MARTY: I don't know which ones --

20 DR. BLANC: What are some of them -- what are
21 the some of them that you know exacerbate asthma? You
22 listed formaldehyde. I think your evidence is, you
23 know, convincingly weak, but other than that, what else
24 do you have of evidence?

25 DR. MARTY: Acrolein, particulate matter,

1 sulphur dioxide, nitrogen dioxide.

2 DR. BLANC: Well, sulphur dioxide is not on the
3 list of --

4 DR. MARTY: Right. Right. But what I'm saying
5 is there are air pollutants out there for which there
6 are good evidence that they're associated with the
7 exacerbations of asthma.

8 The other hypotheticals about agents that would
9 interfere with glucose metabolism, I don't know if there
10 are chemicals out there that do that that were listed as
11 TACs. So, you know it's --

12 DR. BLANC: Again, it's theoretical. I'm just
13 trying to get the examples on the table so I can
14 understand all the thinking process. That's all I'm
15 trying to get at.

16 CHAIRMAN FROINES: Well, I think the other
17 point -- the other point that I'm trying to get to is I
18 think in the end we want a document that lays out the
19 criteria quite explicitly, and here we have a very
20 specific criteria, which is the exacerbation of asthma,
21 and that's associated with small airways and so forth.
22 And you're not including most irritants necessarily in
23 that criteria.

24 So we just need -- when we finally get a full
25 document that those criteria become very well defined so

1 that everybody who reads the document knows exactly what
2 the basis of the decision making was.

3 DR. MARTY: Okay.

4 DR. BLANC: And, you know, the problem with --
5 you don't want to over weight asthma because it's easy
6 to diagnose and the hospitalization rates are clear
7 since no one gets hospitalized for having lost five
8 points of their IQ, you know, due to a chronic
9 neuro-toxicant. So, you know, there's that issue also
10 to be contended with.

11 And I think you're going to need to be very
12 explicit that, you know, using this criterion for this
13 health endpoint doesn't mean that there are other health
14 endpoints, which are probably a great deal more -- well,
15 that could be at least as serious if not more serious.

16 You allude to those as though they're not
17 there, but you do talk about them.

18 CHAIRMAN FROINES: Paul, do you want to raise
19 any questions about criteria at this point, or do you
20 want to save it until we go through the chemicals?

21 DR. BLANC: You mean criteria for how --
22 generic criteria other than the asthma?

23 CHAIRMAN FROINES: Yeah.

24 DR. BLANC: I think we talked about to an
25 extent earlier the issue of things which are -- have

1 neurotoxicologic mechanisms in general for the CNS and
2 their implications generically for the developing
3 organism, and I think that that's an area in which your
4 criteria also need to be explicit.

5 It may not require the same degree of -- well,
6 you should spell out what kind of evidence-based
7 criteria you would need or not need because there
8 you're -- the argument is so direct and so biologically
9 obvious that things which are CNS neurotoxins are going
10 to differentially impact the developing nervous system
11 of an infant.

12 DR. MARTY: We did in the introduction go
13 through several organ systems that we thought were
14 critical.

15 DR. BLANC: I know. I know. I know. I'm just
16 saying.

17 DR. MARTY: It's not spelled out enough,
18 though, is what I'm hearing.

19 DR. BLANC: Well, I think that later on --
20 because that's buried in a whole generic discussion
21 about theoretical ways in which -- in which children
22 could be at risk but it's not -- it's never translated
23 into, therefore, what kind of information we would be
24 looking for from human studies or from animal studies
25 that would support an effect.

1 I think that there's another area of the
2 document that's a bit murky in terms of what your
3 thinking was. If you want us exposed to something which
4 is a clear teratogen, and then an infant is born without
5 legs and then has a normal, legless life span, your
6 argument is not that -- or is it, that being legless in
7 childhood has a differential impact on your childhood as
8 compared to your adulthood?

9 DR. MARTY: The argument would be that if you
10 had that exposure as an adult, you couldn't possibly
11 have that effect. That's the argument.

12 DR. BLANC: But you're not even a child yet.
13 You're in utero. So why are children -- the law has to
14 do with infants and children, not with fetuses. So at
15 what point -- I'm not trying to make an argument here.
16 I'm certainly not trying to make any kind of, you know,
17 backdoor, you know, discussion about, you know, when
18 life begins, et cetera, but I'm trying to understand
19 your thinking.

20 And when -- what you consider a child in terms
21 of -- or an infant and what your criteria for
22 considering pre-term exposures are in terms of
23 susceptibility, because there was a subtext in this
24 document which seemed to imply that you considered any
25 pre-term exposure for which there was fetal

1 susceptibility as being evidence of childhood --
2 childhood sensitivity.

3 And did you, in fact, have your legal counsel
4 comment to you on whether or not that was within the
5 scope of the law as written, since the law does not
6 mention fetuses or prenatal exposure?

7 DR. MARTY: I think it's impossible to argue
8 that developmental toxicity does not impact infants and
9 children differentially.

10 DR. BLANC: Yeah. But over adults?

11 DR. MARTY: Because if you get the exposure as
12 an adult, you don't have the developmental toxicity.

13 DR. BLANC: But they haven't gotten it as a
14 child either. They've gotten it as a fetus.

15 DR. MARTY: I don't think that maturation of
16 the organ system cares whether it happens in utero or
17 postnatally. If the impacts are because the organ
18 system is maturing, that doesn't occur when you're an
19 adult. Then there's the argument for differential
20 impacts.

21 I would like to point out that we don't think
22 that all developmental toxins should automatically be on
23 the list because there's exposure consideration and how
24 potent it is as a developmental toxicant.

25 DR. BLANC: But let's say, theoretically,

1 thalidomide were an air pollutant. You would say it
2 should be on this list; correct? I'm just trying to
3 understand.

4 DR. MARTY: If there was exposure and so forth
5 and so on.

6 DR. BLANC: Yeah. If it was an air pollutant,
7 if there was a toxic air pollutant called --

8 DR. MARTY: It should be considered.

9 DR. BLANC: And based on the available
10 evidence, it would -- assuming that there was exposure,
11 it would be --

12 DR. MARTY: It should be considered for
13 listing.

14 DR. BLANC: Based on that effect.

15 DR. MARTY: Right.

16 DR. BLANC: I'm just trying to understand
17 your -- I think that you need to be even more explicit
18 than you are that in fact an isolated teratogen would be
19 considered because -- for the argument that you just
20 made, if that's the argument you want to make. I'm not
21 sure that that was the intent or not the intent of the
22 legislation, but if that's your interpretation of it, at
23 least you should be explicit about it, even more
24 explicit.

25 I mean, it is there because it keeps coming up

1 in your rationale for considering things.

2 DR. FUCALORO: Just thinking it through, again,

3 I know alcohol is not an air pollutant that we're

4 talking about but a mother using alcohol would then

5 be -- if that were an air pollutant, wouldn't that be --

6 fall under the same category as thalidomide?

7 DR. BLANC: By their --

8 DR. FUCALORO: By their definition.

9 DR. BLANC: Yeah. I assume so.

10 DR. FUCALORO: Of course I -- so something that

11 a mother is exposed to that the kid may -- that a

12 newborn may not be exposed to, isn't that where we're

13 going?

14 CHAIRMAN FROINES: No. That's not the issue.

15 DR. BLANC: They're considering in utero

16 exposure.

17 DR. FUCALORO: But there are some things a

18 mother is exposed to that can damage the uterus in some

19 fashion -- rather the fetus in some fashion, but her

20 child, a newborn, may not be exposed. It would also be

21 considered in this group.

22 DR. BLANC: That's what they're saying.

23 DR. FUCALORO: That's what you're getting at.

24 Yeah. I think it has to be thought through. Is that

25 what you mean? That's what you're asking.

1 DR. BLANC: That's what I'm asking, and their
2 answer is yes.

3 CHAIRMAN FROINES: You realize that also, I
4 mean, Congress, the House of Representatives, just
5 passed a law yesterday --

6 DR. FUCALORO: That's right.

7 CHAIRMAN FROINES: -- mandating that damage to
8 the fetus was considered an illegal act.

9 DR. FUCALORO: Well --

10 CHAIRMAN FROINES: And so it obviously has
11 implications for issues of choice, so that this --
12 taking this position isn't trivial as a matter of public
13 policy.

14 DR. FUCALORO: Yeah. That's why I mentioned
15 it.

16 DR. BLANC: See, for lead, it's not an issue.
17 For mercury it's not an issue because whether or not
18 there would be -- clearly there are effects in utero,
19 but there are clearly effects to neonates, so that's not
20 an issue.

21 Carbon monoxide is not an issue because yes,
22 there's even more hemoglobin in your fetus, but there's
23 still an awful lot of fetal hemoglobin when you're a
24 very early neonate. That's not an issue. But for some
25 of the things you're talking about where the evidence is

1 just teratogenic toxicity, then you're really making the
2 argument, and we're not really talking about central
3 nervous system sensitivity which goes on for months and
4 years of childhood, then, you know, the DES kind of
5 argument, which you're very explicit about in your
6 introduction.

7 And I think it's a bit of a murky area, in
8 fact. I'm not sure that I -- I'm not sure that I accept
9 your argument logically that it's -- that it logically
10 flows, that that means -- that that is the same as
11 having newborn or childhood sensitivity or
12 susceptibility because the adult -- I mean, the damaged
13 child who survives to adulthood -- the fetus has
14 survived to infancy and childhood and then to adulthood,
15 but the susceptibility is not in childhood or in
16 infancy.

17 And the whole piece of legislation, as I read
18 it, never uses the word "fetus" or "fetal" or "prenatal"
19 anywhere.

20 DR. MARTY: Well, let me just give you another
21 example. George is kicking me. But things that result
22 in --

23 DR. FUCALORO: Excuse me. Why is he kicking
24 you?

25 DR. BLANC: That's between them.

1 DR. MARTY: Because he wants me to stop. If
2 something impacts birth weight, for example -- birth
3 weight is a good example. Low birth weight babies,
4 there's a linear relationship between birth weight and
5 infant mortality, and infant mortality I consider an
6 effect that occurs only if you're an infant.

7 DR. FUCALORO: Do you mean linear, or do you
8 mean there is a relationship?

9 DR. MARTY: It's pretty much linear. It's
10 pretty much linear. And a lot of low birth weights, you
11 know, generalize chemical stress. I think that's an
12 important issue. If there's chemicals out there that do
13 this, and you're breathing them, that's an impact.

14 DR. BLANC: Like it --

15 DR. MARTY: It's just -- it's different than
16 terata. It's different than the production of terata.

17 DR. ALEXEEFF: Another point to make on this,
18 other than the fact that I wasn't kicking her, is
19 that --

20 DR. FUCALORO: It was accidental; right?

21 DR. ALEXEEFF: No. Is that the way -- and we
22 could add this type of information to the document. The
23 way we reviewed developmental toxicity, there's not
24 complete concurrence in the effects. In other words, we
25 can do an animal model, and we might get some effect,

1 but we're not sure if in humans it will be expressed as,
2 for example, low birth weight. A lot of it has to do
3 with the timing of the doses and the species that's used
4 and some other factors.

5 The other issue is that since development
6 doesn't stop at birth, or if there's early birth, then
7 there's still development processes happening. And if
8 all we have is the data of exposure in utero, we have
9 to -- we look at that to see if, you know, it's likely
10 to affect what's occurred.

11 Although, you mentioned thalidomide, that's not
12 the only effect it has. It does have some neurotoxic
13 effects as well. So we might -- I think what we'll have
14 to do is we'll have to flesh out a few more reasons as
15 to what we're taking into account, and I don't think we
16 have, necessarily, an example where there is clear
17 teratogenicity in utero, and postnatally it's clear
18 there's no differential effect in children. I don't
19 think we have that kind of example.

20 It's more like --

21 DR. BLANC: Well, I think --

22 DR. ALEXEEFF: There's little evidence. And
23 what evidence shows that there's this differential
24 effect between mother and fetus, and the rest of it is
25 all, you know, less clear. And the concern is that, you

1 know, development continues, and they would be more
2 susceptible.

3 DR. BLANC: Well, I think to the extent that
4 you're talking about -- and you're a pediatrician, so I
5 think you should respond to this perhaps. But to the
6 extent that you've identified in utero effects, which
7 are particularly notable in the last trimester, and
8 you're certainly on much firmer ground to make some
9 assumptions that, in fact, there would also be effects
10 if newborns were exposed.

11 But to the extent that you're dealing with
12 teratogenic effects, which require them to be fairly
13 early in gestation, then I think you're much less able
14 to make the kind of leap that you're making.

15 Let me ask another hierarch question: If you
16 had two substances, one of which you had convincing data
17 that could aggravate asthma and another which you had
18 fairly convincing data that it could initiate asthma, in
19 your hierarchy of issues, as I read your document, that
20 substance which could tend to initiate asthma would be
21 far more important; is that correct?

22 DR. MARTY: I'm not sure it would be far more
23 important.

24 DR. BLANC: But it --

25 DR. MARTY: But it would definitely be

1 important. That's an important issue.

2 DR. BLANC: It's an irreversible effect, isn't
3 it?

4 DR. MARTY: Yes, it is.

5 DR. BLANC: And then you said irreversible
6 effects are more important than reversible effects?

7 DR. MARTY: Yes. Those have an important
8 effect on your immune system. Those are important
9 issues.

10 DR. BLANC: And in terms of something that
11 could aggravate asthma versus something which could
12 cause neural, developmental impairment, literally, the
13 neurotoxin would be more important. It just doesn't
14 effect.

15 DR. MARTY: You know, those kinds of issues are
16 extremely difficult. Those questions are hard to
17 answer.

18 DR. FUCALORO: But you have to answer them.

19 DR. MARTY: The prevalence of asthma is huge.
20 There are so many people with asthma. You are impacting
21 a lot of people when you have things that exacerbate
22 asthma in the air. The neurotoxicants probably impact
23 fewer people. But if I had my choice, I think I'd
24 rather have asthma than developmental neurotoxicity. I
25 mean, that's about all you can do to weight that kind of

1 an issue.

2 DR. FRIEDMAN: But even there, the question of
3 aggravating asthma versus causing it, if it only caused
4 it in one in 10,000 people exposed, but it aggravated
5 severely all the people who had asthma, I think then the
6 aggravation would be worse than the causation.

7 DR. BLANC: Well, I was actually asking the
8 question in a simpler format. I wasn't -- if you
9 assumed, I wasn't taking prevalence as the issue.

10 DR. FRIEDMAN: But you can't ignore prevalence.

11 DR. BLANC: No. You could take that as a
12 separate weighting issue because in your document you
13 talk about things that a matter, reversibility versus
14 irreversibility. There's no real hierarchy that one can
15 follow in terms of, you know, what matters. Clearly
16 prevalence is one weighting. You have a lot of
17 different things.

18 DR. MARTY: People have tried to develop such
19 hierarchies. The U.S. EPA tried for years to develop
20 hierarchy. Are you going to call a carcinogen worse
21 than a neuro-toxicant and so forth? And they were
22 unsuccessful. They just gave up. And it's just -- it's
23 so difficult. Are you going to put a "No. 1" on
24 carcinogens and No. 2 on -- you know, it's just a
25 balancing act. You have to think about all kinds of

1 other issues that come into play, which is what we tried
2 to do.

3 CHAIRMAN FROINES: Tony.

4 DR. FUCALORO: Yeah. Well, you certainly did
5 some quantitative ranking using the scale you did in
6 No. 2 and 3 at the beginning of this document relating
7 to toxicity and exposure.

8 DR. MARTY: Yes.

9 DR. FUCALORO: Now, I recall a couple years ago
10 when you brought before us the methodology you used to
11 decide which chemicals you would investigate as a TAC,
12 and you have a methodology which listed. And I thought
13 it was a very good document. I don't have it any
14 longer, unfortunately. I didn't bring it with me, if I
15 do. But have you looked at that methodology?

16 DR. MARTY: Yes.

17 DR. FUCALORO: It seems to me this would be --
18 that would be a good start. Maybe you have already.

19 DR. MARTY: It actually was the starting point.

20 DR. FUCALORO: It actually was the starting
21 point. All right. That's fine.

22 CHAIRMAN FROINES: I don't know how we are to
23 resolve the issue of the in utero toxicity because
24 Melanie knows that I feel the way Paul feels, and we've
25 heard from Paul. So at least two of us have strong

1 reservations about the thalidomide example as an example
2 of differential susceptibility, and I don't quite now
3 how to resolve the issue. But it's a very troubling one
4 and clearly has much broader policy implications to the
5 degree that one accepts the current definition.

6 So I'm at a -- George and Melanie, I'm at a
7 loss for how to proceed on that one. I suspect that one
8 ought to get some legal counsel to ask something about
9 the intent of the legislature with respect to children.
10 I don't know whether -- I suspect we will not get any
11 kind of answer that will be very definitive but --

12 DR. MARTY: We could --

13 CHAIRMAN FROINES: -- I don't have -- I mean,
14 clearly we have a clear difference of opinion here that
15 at least two of us hold relative to your point of view.
16 And we haven't sort of polled the rest of the panel, but
17 I don't know how to resolve it.

18 DR. BLANC: I think the first step is to have
19 it explicated clearly in the document, because,
20 actually, I can't really respond to it because it's not
21 there for me -- there's no there there for me to respond
22 to it subtextually. So you need to firmly elucidate
23 what it is you're trying to do in that regard and
24 then --

25 DR. MARTY: Then we can talk about it. And it

1 also helps to go through specific examples of the
2 chemicals.

3 DR. FUCALORO: To the extent possible -- I
4 think John's point is correct. I think you should try
5 to uncover the original intent of the legislature, but
6 to the extent -- I don't know if that's -- certainly
7 they're still around, unlike the American founders,
8 but -- so you might have a real chance to get some
9 direction from them.

10 CHAIRMAN FROINES: Well, I also think that,
11 going to the next step, that this is a particularly good
12 discussion, and it illustrates the fact that there are a
13 lot of issues that are really not as well defined in the
14 document as they need to be in the long term. And so
15 the issue of neurotoxicity is certainly one that
16 requires subsequent follow up. This issue is another,
17 and perhaps we'll identify others as we go along.

18 But that when this document is finished,
19 hopefully it will be a document which is only about
20 these issues, and all the other stuff that's in the
21 current documents will be gone, the review of -- the
22 review of the toxicity of the individual chemicals will
23 be gone, and the specific criteria will be laid out, and
24 then the relationship between the criteria and the
25 evidentiary basis for a decision will be clear.

1 DR. BLANC: Actually, let me come back to
2 another example which may help clarify for me what your
3 thinking was. Let's take DES, not thalidomide. Let's
4 say DES was an air pollutant. Where the effect were --
5 the exposure occurs in utero, the differential main
6 toxicity is manifest in adulthood. Therefore, that
7 would be something that you would not consider because
8 it doesn't preferentially effect children at all or
9 because it's -- because the exposure occurred in utero,
10 you would include it?

11 DR. MARTY: We would include it.

12 DR. BLANC: Now, see. I think that's
13 completely bizarre.

14 DR. MARTY: Well, let me explain why. Because
15 DES does not have that impact if you're not a maturing
16 organism. The only reason it's manifested as a teenager
17 is because you were exposed in utero. If you were
18 exposed when you were 16, it wouldn't have manifested
19 the same toxicity.

20 DR. BLANC: But if you were exposed when you
21 were an infant or a child --

22 DR. MARTY: I don't know. No one's done
23 those -- no one's done those experiments with DES.

24 DR. BLANC: But there's no particular reason to
25 think that it wouldn't work. Well, are you sure no

1 one's done that with animals?

2 DR. BYUS: With animals they've done that.

3 DR. MILLER: This is maybe changing it
4 slightly, but from the neurotoxicity standpoint with
5 which you're very interested, there is good evidence for
6 a number of chemicals, particularly metals, that while
7 the most severe effects occur during the earlier stages
8 of development, that, in general, those correlate, if
9 you can study them well enough as has been done with
10 lead, to an effect that is also found in postnatal life.

11 DR. BLANC: No. I agree with that.

12 CHAIRMAN FROINES: We buy that. That's a
13 given.

14 DR. BLANC: I don't think that that applies to
15 the issue of vaginal cancer.

16 DR. MILLER: It doesn't. But what Melanie is
17 saying is true it's certainly a number of areas where we
18 don't know, perhaps, you know about the postnatal
19 exposure because we don't have it. We don't have that
20 to look at. Nobody's done those experiments.

21 CHAIRMAN FROINES: And so if it was an air
22 pollutant -- if DES was an air pollutant and one was
23 exposed to it throughout one's life, does it increase
24 the risk of breast cancer and ovarian cancer because of
25 its estrogenic nature? It could. So we don't really

1 know.

2 DR. MILLER: We don't know.

3 CHAIRMAN FROINES: But one could predict that
4 it has hormonal-related cancer effects.

5 DR. BLANC: I was only asking the question to
6 clarify your thinking, and I think I understand your
7 thinking, which is that if something is a teratogen or
8 has effects that are only -- can only be manifest with
9 exposure in utero, even -- even were the only known
10 effects to be seen in adulthood, you would consider that
11 to be fair game under this legislative mandate.

12 DR. MARTY: We would.

13 DR. BLANC: All right. I understand your
14 thinking.

15 And I would reiterate what John said, which is,
16 A, get some legal counsel, and, B, make it very, very
17 clear. And I think, by the way, that you may find in
18 this document a brief section which uses examples of
19 chemicals for which you're not even -- you're not
20 remotely suggesting that they be addressed here because
21 they either aren't air pollutants or, you know, it
22 doesn't matter.

23 But for illustrative purposes, were they to be
24 air pollutants, why they would have been something you
25 would have looked at very closely would be very helpful.

1 DR. MARTY: Okay.

2 DR. BLANC: You know, radiation, for example,
3 which you alluded to in the introduction.

4 DR. MARTY: Right.

5 CHAIRMAN FROINES: I think that you realize
6 that you may have -- you've had public comments up to
7 now, but a very large number of groups who have concerns
8 about abortion, when they discover that this is in your
9 document, may also have significant concerns, and that
10 it may -- this may be opening a box that we all
11 understand may occur, but it's -- it will -- it changes
12 the nature of the discussion.

13 DR. BLANC: Because you're essentially saying,
14 if I need to be even more explicit, you're saying that
15 fetuses are the same as children or infants.

16 DR. MARTY: Well, we're saying that development
17 starts at conception and goes through birth and out into
18 adolescence, and we're not distinguishing development
19 that occurs before birth with development that occurs
20 after birth.

21 CHAIRMAN FROINES: But then we're going to --
22 then it's possible that when one gets into defining
23 risk- based approaches for those chemicals, you will
24 define -- you will define the risk associated with those
25 events.

1 DR. MILLER: Not -- perhaps this is germane,
2 but just as a point of reference, the field -- the
3 developing field of pediatric environmental health has
4 in general taken development from prior to birth through
5 adolescence as the field, for whatever that's worth.

6 DR. MARTY: And actually in a minute when we
7 talk about benzene, we're going to talk about
8 pre-conceptual parental exposure.

9 CHAIRMAN FROINES: Right. I want to be on the
10 record and say that I think that in utero exposure at
11 various time frames can affect the health outcome of an
12 individual throughout their lifetime, and I think
13 there's increasing evidence to indicate that there is a
14 whole series of health outcomes that may get impacted
15 over a long period of time from in utero exposure. So
16 it's a developing field, but this is a -- this is in
17 relation to this particular law. That's the issue here.

18 DR. MARTY: Should we move on to the individual
19 chemicals?

20 CHAIRMAN FROINES: Please.

21 DR. MARTY: I just thought I would go just in
22 order that they're listed, which is alphabetical,
23 through the first five that we proposed or suggested for
24 listing and then through the remainder.

25 DR. FUCALORO: Is there a handout that covers

1 this?

2 DR. MARTY: The handouts are coming.

3 DR. FUCALORO: Oh. They are.

4 DR. MARTY: That's what Peter was asking me
5 about.

6 Tom McDonald is a toxicologist at OEHHA. Tom
7 is going to give the presentation on benzene. What
8 we're going to try to do is summarize what evidence we
9 considered to implicate a chemical as having a
10 differential effect, and then I will summarize briefly
11 comments we got on those chemicals and our responses,
12 which all of the panel has had the comments and
13 responses sent to them already. So it will be a brief
14 summary.

15 DR. MCDONALD: Well, hello, everyone. The last
16 discourse that this group had will certainly feed right
17 into discussion here on benzene.

18 Benzene was placed in Tier 1 primarily because
19 of suggestive evidence for differential susceptibility
20 with respect to cancer. The evidence summarized in one
21 slide here is the suggestive evidence of associations
22 between parental exposures, both maternal and paternal,
23 and childhood leukemia in some studies but not others,
24 and there is some supportive animal data to support
25 these epidemiological findings.

1 Also, there's a possible increased lifetime
2 cancer risk from early life exposures to benzene
3 relative to adult exposures, and that evidence comes
4 from one set of inhalation studies in animals conducted
5 by Malatoni et al. And there is indirect evidence from
6 other leukemogens, namely radiation, such that we see
7 early life exposures to radiation induce a greater
8 excess of leukemia mortality compared to exposures
9 occurring at, quote, "working age individuals."

10 Next slide, please. Just to briefly summarize
11 that benzene also is considered a developmental
12 toxicant. Benzene was listed as a developmental
13 toxicant under Proposition 65 in 1997. And currently
14 OEHHA is working to develop a maximum allowable daily
15 intake level based on the developmental effects of
16 benzene.

17 Since it is still likely that cancer will drive
18 the regulatory effort --

19 CHAIRMAN FROINES: Can I interrupt? I hope
20 that you guys can avoid doing what you just did. I want
21 to know what is the science with the -- associated with
22 benzene as a developmental toxicant? I don't give two
23 hoots about what OEHHA did under Prop 65. The science
24 is what we're talking about here, not about an agency
25 decision. And so what happens replete throughout this

1 document is references to agency decisions, and I think
2 that that doesn't make an argument that has any weight
3 for me.

4 I want to know what is the scientific basis for
5 a decision, not what did EPA say? What did OEHHA say?
6 What did Joe -- Agency X say? I think that what happens
7 is there gets to be this reliance that says if some
8 agency says something is so, therefore, it must be so.
9 And, as a scientist, I don't accept that whatsoever.

10 DR. MCDONALD: Okay. Just to respond that the
11 developmental evidence was presented as a context that,
12 you know, this is what's available. But the focus of
13 the summary in the original draft was cancer, and I
14 tried to discuss in more detail that evidence, and
15 that's what I will continue to discuss here. There will
16 be no more slides on developmental toxicity of benzene
17 beyond this one.

18 Next slide, please.

19 CHAIRMAN FROINES: Well, is it -- is it a basis
20 for your decision?

21 DR. MCDONALD: No.

22 CHAIRMAN FROINES: No. So we didn't really
23 even need that slide, did we? So why do we have it
24 then?

25 DR. MCDONALD: I was just --

1 DR. MARTY: I think that --

2 CHAIRMAN FROINES: I told Melanie that I wanted
3 to have -- that you come and present the criteria for
4 decision making and the basis for decision making. I
5 don't want you to present information that did not serve
6 as the basis for your decisions because we can't judge
7 that.

8 We have to review what you think is the
9 rationale for the decision, and that has to be what this
10 panel can deal with. We can't deal with things that are
11 not directly relevant to the question before us.

12 DR. MCDONALD: Okay.

13 CHAIRMAN FROINES: And I don't mean to be harsh
14 about it, but we've been here for hours, and we're going
15 to be here for hours and days more. And we have to
16 really focus on the science associated with the decision
17 making process within the context of your criteria.

18 DR. MARTY: Okay. Well, let's do that right
19 now.

20 The evidence for differential susceptibility
21 with early life exposures to benzene, it can be thought
22 of in two categories, if you will. The paternal
23 exposures to benzene and how it might relate to
24 increases in childhood leukemia, as well as early life
25 exposures, either in utero or postnatally that may

1 increase lifetime excess of cancer risk.

2 Next slide, please. With respect to benzene
3 and childhood leukemia, there is suggestive evidence in
4 some epidemiological studies, but not others, both from
5 paternal exposures, that is, exposures to the father
6 pre-conceptually, as well as maternal exposures, thus in
7 utero. And I'd like to stress that this information
8 will -- although suggestive, would be very difficult to
9 establish a causal relationship between these two, you
10 know for childhood leukemia and benzene.

11 I should note that there is some animal
12 evidence that would support such associations, and that
13 includes benzene exposure in vivo, which causes DNA
14 damage to sperm, as well as transplacental genotoxicity,
15 as well as transplacental altered hematopoiesis, which
16 is believed by many to be an important mechanism in
17 benzene to produce carcinogenesis.

18 Next slide, please. And oh. By the way, I
19 have detailed slides of the epidemiological studies at
20 the end if you care to go into those in more detail.

21 Early life exposures to benzene and increased
22 lifetime leukemia risk, there's only one animal study on
23 benzene that has exposed prior to weaning, and that is
24 the Malatoni studies. Offspring that were exposed in
25 utero through lactation and adulthood, that is, a total

1 of a 104-week exposure, resulted in greater incidence
2 of -- relative to the exposures to the dams that were
3 exposed for 85 weeks.

4 So I've shown here Zymbal gland, which is the
5 most consistent tumor site found in both species of
6 rodents commonly tested. You see the treated females
7 from the offspring had a 12 percent tumor rate compared
8 to controls which were zero percent. Whereas, the rate
9 in the dams was 6 percent and the controls were 2
10 percent.

11 So this roughly means that a 20 percent
12 increase in exposure time resulted in a twofold increase
13 in tumor rate. And, as stated in the draft, we need to
14 really do a detailed assessment to see if such an
15 increased tumor rate can be explained by dose or whether
16 there is some suggestion of a differential
17 susceptibility.

18 Next slide, please. With respect to the human
19 evidence in this question of lifetime leukemia risk,
20 there is no direct studies which have looked at early
21 life or childhood benzene exposure and lifetime excess
22 of cancer risk. However, there is age-dependent
23 evidence from other leukemogens. Of course, the biggest
24 data sets are from radiation.

25 And just to note that radiation-induced

1 temporal patterns of leukemia have for decades been used
2 to weight benzene-induced leukemia risk, including the
3 current cancer potency estimate for the California TAC
4 for benzene, and I can explain this in more detail if
5 you'd like.

6 Next slide. If we look at the available
7 evidence from radiation-induced leukemia with respect to
8 age at exposure, we see a differential pattern such that
9 exposures early in childhood cause a greater excess
10 leukemia mortality than exposures occurring, say, during
11 the working age of, say, 20 to 50, and that, of course,
12 is, you know, the ages with which the cancer potency of
13 benzene is based on.

14 And this is a period, you know, suggested by
15 the radiation data of lowest susceptibility to
16 leukemogenesis. So that concludes the core evidence.

17 DR. GLANTZ: This is --

18 DR. BYUS: Mechanistically, I mean, comparing
19 benzene and radiation in terms of the mechanism --

20 DR. MCDONALD: Yeah.

21 DR. BYUS: -- by which it might induce cancer,
22 what do they think about that?

23 DR. MCDONALD: Well, I think it's more just an
24 inherent. It's an inherent -- it's trying to get at the
25 inherent properties of the turnover of bone marrow and

1 the response to bone marrow to DNA damage.

2 DR. BYUS: So there's some similarity in the
3 mechanism?

4 DR. MCDONALD: Yeah. There's lots of
5 comparative data. For example, after radiation
6 exposure, excess leukemia rises quite rapidly within
7 five to ten years following exposure, and then, unlike
8 other cancers, comes back to background rates by about
9 30 years following exposure. Now, that is very
10 consistent with several classes of chemotherapeutic
11 agents as well as consistent with what we see in
12 benzene-exposed leukemia cohorts from benzene-exposed
13 workers.

14 So there is lots of data to suggest very
15 similar temporal patterns between the two responses
16 between these two types, chemical versus radiation. So
17 I think it's a reasonable -- biologically, it's a
18 reasonable argument to make.

19 DR. GLANTZ: But -- well, that was actually --
20 I was very confused by that, too, and, I mean, were you
21 saying in the document that benzene -- that there was
22 some interaction between benzene and radiation exposure,
23 or were you just saying that you think that benzene
24 exposure behaves, in terms of effects on risks, behaves
25 similarly to radiation?

1 DR. MARTY: That has been the pattern with
2 other analysis of temporal responses, yes.

3 DR. GLANTZ: And could you explain again why
4 you would expect that to be the case? What's the
5 affirmative evidence that benzene exposure should act
6 like radiation exposure?

7 DR. MCDONALD: Well -- sure.

8 DR. MARTY: I think what we're trying to say is
9 that other known leukemogens, including chemotherapeutic
10 agents and radiation, exhibit this wavelike pattern of
11 susceptibility to leukemia, and that that points to
12 something innate about the hematopoietic system in terms
13 of its sensitivity to leukemogens at those various ages.

14 DR. GLANTZ: I see.

15 DR. MARTY: If that holds true for benzene,
16 then you would expect that for benzene.

17 DR. BYUS: I still find, you know --

18 DR. GLANTZ: Yeah. I think that's --

19 DR. BYUS: I could still see chemotherapy and
20 radiation causing DNA damage directly, mutation. It's
21 hard to see that for benzene mechanistically. But I
22 see -- I understand what you're saying about the
23 turnover of the marrow and --

24 DR. MARTY: It's genotoxic metabolites of
25 benzene.

1 DR. BYUS: All right.

2 DR. MARTY: Yes.

3 DR. BYUS: Okay. So there are genotoxic

4 metabolites in --

5 DR. MCDONALD: Yes. Benzene is a very strong

6 clastogen.

7 DR. BYUS: That's the answer.

8 DR. MCDONALD: Yeah.

9 DR. BLANC: But, in fact, the document --

10 the -- I mean, I might have missed this, but in the

11 section on benzene itself, is the analogy with the post

12 chemotherapy incidence of stem cell malignancy, bone

13 marrow malignancy in terms of dose response for children

14 treated for malignancy versus dose response for adults

15 treated for malignancy explicated in the text of the

16 document. The radiation stuff is there.

17 DR. MCDONALD: Yes. There are several

18 published studies describing this temporal pattern.

19 DR. BLANC: There's two temporal patterns

20 you're describing.

21 DR. MCDONALD: Yes.

22 DR. BLANC: I'm not arguing about the -- the

23 germane issue is not the temporal pattern.

24 DR. MCDONALD: Correct.

25 DR. BLANC: There's an increase in incidence

1 five to ten years afterwards where some falls off,
2 because that's true for anyone at any age.

3 DR. MCDONALD: Yes.

4 DR. BLANC: But is there data that shows that
5 per milligram -- per square meter of exposure to -- it
6 says "platinum."

7 DR. MCDONALD: Yeah. I'm not aware of such
8 data, and such data would be complicated by the fact
9 that children often are given, I believe, higher doses
10 of chemotherapeutic agents because they can tolerate
11 them.

12 DR. BLANC: Yeah. That's what I said. I'm not
13 an expert.

14 DR. MCDONALD: I'm not aware of an analysis
15 that shows increased response to chemotherapeutics by
16 age. There may be.

17 DR. BLANC: Is there?

18 DR. FUCALORO: Can I just make a small,
19 technical point? Your unit risk factor in the benzene
20 report is probably wrong by a factor of two. I think.
21 Compare it with some of the other data. Unless your
22 table is wrong.

23 DR. MCDONALD: Which?

24 DR. FUCALORO: I think you recorded CCL 4 as
25 carbon tetrachloride. I know it's off point, and I'm

1 sorry, Mr. Chairman, but I'm trying to do some
2 calculations, and I want to use the right number.

3 DR. MCDONALD: Well, the unit risk factor in
4 inverse micrograms per meter cubed is 2.9 times 10 to
5 the minus 5.

6 DR. FUCALORO: That's what you have in the
7 document.

8 DR. MCDONALD: That's correct.

9 DR. FUCALORO: Yeah. And in the table it's
10 5.9.

11 DR. MCDONALD: Okay. We'll --

12 DR. FUCALORO: So one of those are wrong.
13 Maybe both of them are. I like to open up all
14 possibilities.

15 DR. BLANC: Let me just follow up on my
16 previous question. The fact that there's a technical
17 response to things which cause leukemia that's much
18 shorter latency than -- for most other forms of cancer
19 is irrelevant to the discussion here. That bears no
20 relevancy at all to the issue of childhood
21 susceptibility, does it? Or did I miss something?

22 The only issue is whether the children would be
23 more sensitive or more responsive to an equivalent dose
24 of leukemogenic agent.

25 DR. MCDONALD: Right. We're just trying to get

1 at some picture of the inherent response of the bone
2 marrow and the only --

3 DR. BLANC: Again, but the first point has no
4 relevance to our argument here.

5 DR. MCDONALD: Well, I've shown age-specific
6 data on radiation. Did I miss something?

7 DR. MARTY: I don't understand the question.

8 DR. BLANC: There are two temporal issues. One
9 is that, yes, it is true that things which cause
10 leukemia tend to have a shorter latency, and then you
11 have a fall off to background levels.

12 DR. MARTY: Right.

13 DR. BLANC: That has no relevancy to our
14 discussion here.

15 DR. MARTY: Correct.

16 DR. BLANC: What has relevancy to our
17 discussion here is if you exposed a three year old to
18 one rad of radiation, would they have a greater
19 incidence of leukemia than a 20 year old exposed to one
20 rad of radiation?

21 DR. MARTY: Yes.

22 DR. BLANC: And then I asked the question, is
23 there similar data for chemotherapeutic agents, and the
24 answer I got was no, not that you're aware of.

25 DR. MCDONALD: Correct. But that -- yeah.

1 DR. MARTY: We're going to look at that because
2 I was under the impression that there are.

3 DR. MCDONALD: I'm just not aware of them.

4 DR. MARTY: It's the most common, secondary
5 cancer following treatment in childhood for other
6 cancers. Whether there's data showing on a per
7 milligram, per kilogram body weight basis, we can dig
8 around for that, but I am remembering that there are
9 those data, so we can look at that.

10 CHAIRMAN FROINES: Am I correct to assume that
11 we've heard the basis for the decision or -- which is a
12 series of articles -- a series of sort of arguments that
13 are --

14 DR. MCDONALD: Yes.

15 CHAIRMAN FROINES: -- somewhat indirect, or is
16 there coming a more definitive statement?

17 DR. MARTY: We have some slides on the epi
18 studies that indicated parental exposure that may be
19 associated with leukemia risks. But that you pretty
20 much have heard the two points.

21 DR. MCDONALD: Right. If you want me to go
22 into details about the epidemiological studies of
23 parents and childhood leukemia, then we can go into the
24 specifics.

25 CHAIRMAN FROINES: I --

1 DR. MARTY: Let's go through them

2 DR. MCDONALD: Would you like to go through it?

3 CHAIRMAN FROINES: I don't know what the panel

4 would like.

5 DR. BLANC: Maybe what we could do is hold that

6 in abeyance and come back to it because I think we need

7 to have some sense of the substances, one as opposed to

8 the other, and it's already a quarter to 3:00. And we

9 do have those on your -- there on your handout --

10 DR. MCDONALD: Yeah.

11 DR. BLANC: -- so we can come back to them

12 without seeing the slides, if we wanted to then at that

13 point to compare --

14 DR. MCDONALD: Whatever the panel would like.

15 DR. BLANC: Mr. Chair, would that be okay?

16 CHAIRMAN FROINES: Yes. I think -- from my

17 standpoint, I think the evidence is extremely weak for

18 benzene at this point given these arguments.

19 DR. BLANC: Well, can we just hear some of the

20 others? Let's get some comparison. I know you're put

21 in the position where you have to name five things. So

22 it may be that this is very weak data. Obviously you

23 felt the data were even weaker for one of the others,

24 but let us just get a sense of where you're coming from.

25 For the group, it's very important I think --

1 DR. MARTY: Okay.

2 DR. BLANC: -- to get comparative cases.

3 DR. MARTY: Okay. Do you want me to hold off

4 on the comments and responses --

5 CHAIRMAN FROINES: Yeah.

6 DR. MARTY: -- on benzene?

7 CHAIRMAN FROINES: Let me ask the panel about

8 that. I asked Melanie if she would be prepared to

9 address comments because for most of us the comments are

10 extremely important. So she was prepared to respond --

11 to give a response to comments. And so the question is,

12 should we move on at this point and take on some other

13 chemicals --

14 DR. GLANTZ: Yeah. I think -- I think --

15 CHAIRMAN FROINES: Or would you like to hear

16 the comments -- the response to comments?

17 DR. GLANTZ: I agree with Paul. I think it

18 would be really helpful to go through the other

19 chemicals, or at least some of them, and then we can

20 come back if there's time and deal with the comments.

21 CHAIRMAN FROINES: Okay.

22 DR. GLANTZ: Because we -- we read them.

23 DR. MARTY: You read them. Okay.

24 DR. GLANTZ: Or at least I read them. I don't

25 know about everybody else.

1 DR. MARTY: Let's go to formaldehyde. Stan.
2 Andy, we're going to go to formaldehyde.
3 DR. DAWSON: Good afternoon.
4 DR. GLANTZ: Why are you quaking?
5 DR. DAWSON: Why am I -- well, after the little
6 interchange. I'm here to defend formaldehyde.
7 DR. GLANTZ: We're very nice.
8 DR. DAWSON: Formaldehyde was chosen for Tier 1
9 based on chronic respiratory response or effects,
10 including allergic effects. It has the potential to
11 exacerbate asthma, and you can see measured impacts on
12 lung function in children, chronic respiratory response.
13 Some indication that children may be more
14 sensitive to lung function changes than adults at low
15 level exposures and carcinogenicity is a concern.
16 Actually, just as an overview of the one study,
17 this study here compares disease response of children
18 and adults directly. Three other studies support this
19 one, suggesting an effect of formaldehyde at even lower
20 exposures.
21 CHAIRMAN FROINES: This is the only one that
22 looks like it has a differential; correct?
23 DR. DAWSON: Yes, this is the only one.
24 CHAIRMAN FROINES: The other three don't do
25 that.

1 DR. DAWSON: That's right.

2 So this is the Krzyzanowski et al., with
3 Quackenboss and Mike Lebowitz. Chronic respiratory
4 effects of indoor formaldehyde exposure, chronic
5 respiratory symptoms were reported and diagnosed. This
6 first slide is just a description of this study. And
7 lung function was obtained by PEFr, peak expiratory flow
8 rate.

9 There was information on tobacco education and
10 NO2 in almost 300 children, 600 adults in 200
11 households, age 5 to 15 years, carried out in Tucson,
12 Arizona. And the mean for formaldehyde is 26 ppb. And
13 they study grouped individuals by less than 40 --
14 between 40 and 60 and above 60.

15 Results: First of all, the disease and
16 symptoms, prevalence of asthma and bronchitis -- chronic
17 bronchitis was significantly greater for formaldehyde
18 above 60 ppb. This is a patent disease now, and P
19 values there were much more significant for the chronic
20 bronchitis than for the asthma. And the kitchen levels
21 of formaldehyde bore the closest fit.

22 The reported symptoms of the children from the
23 questionnaires were not related to formaldehyde. And
24 there are a bunch of symptoms that were asked for, and
25 neither symptoms nor actual disease were significant.

1 That is doctor-diagnosed disease were significant for
2 adults. Yet there was a higher end, remember, in the
3 adults. So we should have seen more power to see an
4 effect.

5 Next, the results for the peak expiratory flow
6 rate which is a measure of general lung function. My
7 understanding it's not just the airway size themselves.
8 It also includes the compliance of the lung. The a.m.
9 and p.m. PEFs declined linearly.

10 DR. MARTY: That's morning and afternoon. They
11 tested at four time points during the day.

12 DR. DAWSON: Yes. And it was equivalent to a
13 22 percent decline at 50 ppb, and that was just
14 significant. The PEFs declined only in the a.m. in
15 adults, and there was a very much smaller effect. And
16 this study did control, to a good degree, for the effect
17 of possible confounders.

18 Next. The next study, which overlaps somewhat
19 but was only on children, was Garrett et al., the
20 increased risk of allergy in children due to
21 formaldehyde exposure in homes. It measured atopy,
22 asthma and respiratory symptoms; eighty children,
23 fifty-three of whom were asthmatic in 43 households.
24 Mean age around 10 years, range 7 to 14 years. This was
25 in a coal mining town in -- fairly near two different

1 mines in Victoria, Australia.

2 Median: Formaldehyde is 12.6 ppb with a
3 maximum of around 100, and again there were three
4 exposure categories.

5 The results: There was a significant increase
6 in the adjusted odds ratio for atopy. 1.4 was the ratio
7 per 8 ppb increase in formaldehyde level. There was
8 more severe sensitization with formaldehyde increase as
9 well. There was no significant increase in adjusted
10 odds ratio for asthma or respiratory symptoms, but they
11 were more frequent in children with higher exposures.
12 And the adjustment was for parental asthma status.

13 DR. FRIEDMAN: Did they look for any other
14 possible parental confounders like parental smoking or
15 socioeconomic status?

16 DR. MARTY: I'm pretty sure they looked at
17 parental smoking. I don't recall anything about
18 socioeconomic. Presumably, it would be relative in a
19 coal town. I assume it would be relatively whole in
20 that respect.

21 Another supporting study is Franklin et al.
22 This is raised, exhaled NO in healthy children is
23 associated with domestic formaldehyde levels. Exhaled
24 nitric oxide for lower airway inflammation is a marker
25 for lower airway inflammation. They also did spirometry

1 and skin prick.

2 There were 200 healthy children, age 6 to 13.
3 This is in Perth, Australia, the other side of the
4 continent, and they divided formaldehyde into two groups
5 at 50 ppb.

6 The exhaled formaldehyde was greater. This is
7 the results. I'm sorry. Exhaled NO is greater in homes
8 with the formaldehyde greater than ppb, and the
9 measurement, just NO, was 16 versus 9 ppb. This is
10 significant after controlling for all other variables
11 and regression at quite a significant level, .002, and
12 this was found to be independent of atopy.

13 Wantke et al., another supporting study,
14 "Exposure to gaseous formaldehyde induces IGE mediated
15 in sensitization in formaldehyde in school children."
16 Specific IGE by rast and symptoms were looked at. Sixty
17 children in three classes before and after a move of the
18 classrooms from a higher level to a lower level of
19 formaldehyde. Mean age was very close to eight years.
20 All the kids were very close to eight years, one grade
21 level, in Vienna, Austria.

22 And notice the formaldehyde levels here in the
23 one class that went from 75 down to 29 and 69 to 23 and
24 43 to 26, so they were down by a factor of three or two.

25 Results.

1 DR. GLANTZ: If you could just -- one thing I
2 don't understand there is when you say formaldehyde
3 exposure -- if you back up one slide -- increased
4 sensitization to formaldehyde, I don't -- so are you
5 saying if they're exposed to formaldehyde once, then
6 they become more sensitive to formaldehyde on subsequent
7 exposures? Is that what that means?

8 DR. DAWSON: In the title?

9 DR. GLANTZ: Yeah. I don't quite understand
10 what you're saying.

11 DR. DAWSON: Exposure to gases induces IGE.

12 DR. GLANTZ: Well, is that what you're saying
13 happens?

14 DR. DAWSON: This is the author's title,
15 "Exposure to Gaseous Formaldehyde Induces IGE Mediated
16 in Sensitization." That's what their claim is.

17 DR. GLANTZ: So you're --

18 CHAIRMAN FROINES: He's asking what that means.

19 DR. GLANTZ: Yeah.

20 DR. MARTY: I think that the reason we're
21 concerned about that is that, typically, people have
22 thought of formaldehyde sensitization as occurring at
23 high occupational exposures, and, therefore, it's really
24 an adult problem, not a child problem.

25 And this paper is measuring

1 formaldehyde-specific IGE in kids who were exposed at
2 commonly encountered indoor air levels. That to me was
3 significant because it kind of bucks the tide of this
4 idea that you have to have real high exposures to find
5 any evidence of sensitization. Whether it's clinically
6 different or not is a different issue.

7 DR. GLANTZ: But the question I'm just asking,
8 when you talk about sensitization, is that saying that
9 you get sensitized -- you get exposed to formaldehyde,
10 that sensitizes you so the next time you're exposed to
11 formaldehyde, you get a bigger effect? Or are you
12 saying -- is this a measure -- are you just saying that
13 these kids were responsive to low doses of formaldehyde?

14 DR. MARTY: It's the latter. We're saying they
15 were responsive to low doses. We're not sure if you
16 took these kids and gave them various exposures how --

17 DR. BYUS: It's the classic sensitization to
18 make IGE after the first exposure such that when they're
19 exposed again, there's the antibody there, and it binds
20 to it and gives you the massive response.

21 DR. GLANTZ: Okay.

22 CHAIRMAN FROINES: I assume this is just a
23 cross-sectional study where they took a population of
24 kids, measured their IGE and measured their formaldehyde
25 levels.

1 DR. DAWSON: Right.

2 DR. MARTY: It was specific kids in a school
3 district in Vienna, and they were interested in it
4 because the reason they moved the kids was because they
5 had high concentrations of formaldehyde, and they were
6 all in these little rooms with paneled particle board,
7 and then they moved them out to a different set of
8 classrooms and took the opportunity of measuring the IGE
9 when they --

10 DR. DAWSON: Of course they came to the --

11 DR. BLANC: But their IGE should have stayed
12 the same, virtually.

13 DR. MARTY: Well, I think the -- the IGE
14 dropped after the children were moved to a lower
15 formaldehyde concentration.

16 DR. BLANC: It's not clear to me that it would
17 have. Why would it have dropped?

18 DR. DAWSON: Well, I think that --

19 DR. BLANC: Your symptoms may drop, which they
20 didn't.

21 DR. DAWSON: Yeah.

22 DR. BLANC: Well, none of that -- I mean, this
23 isn't particularly relevant to children being more
24 likely to become more sensitized than adults, of course.
25 But perhaps we can go back to -- just a question about

1 the main study that drove all of this is the
2 Krzyzanowski study?

3 DR. MARTY: Right.

4 DR. BLANC: That's -- everything else is
5 ancillary, supportive in your view.

6 DR. DAWSON: Right.

7 DR. MARTY: Supporting that you can measure
8 formaldehyde respiratory health impacts at low levels,
9 that you can find formaldehyde-specific IGE even at low
10 levels in kids. There is not -- there were not
11 comparisons to adults in these other studies.

12 DR. BLANC: So in the Krzyzanowski study, the
13 linear relationship cross-sectionally between peak
14 expiratory flow and the measured formaldehyde levels, in
15 your slide where you say there was a linear decline, I
16 haven't gone back to read the article myself. I'm just
17 trying to understand what you were trying to say.

18 There was a dose response relationship cross-
19 sectionally between peak flow in all children as a
20 group, which included some subset of them that had
21 asthma or didn't have asthma.

22 DR. DAWSON: Right.

23 DR. BLANC: So it wasn't a study that looked at
24 whether children with asthma were more responsive to
25 formaldehyde.

1 DR. MARTY: Correct. That's right.

2 DR. BLANC: So, in fact, it really is just a
3 study of the irritant effects of formaldehyde insofar as
4 they're just looking at -- if that's, in fact, the
5 explanation of the cross-sectional relationship that we
6 see.

7 DR. MARTY: Yes. It -- yes.

8 DR. BLANC: So you don't have data that shows
9 that asthmatics exposed to formaldehyde have a bigger
10 response than non-asthmatics.

11 DR. MARTY: That's right.

12 DR. DAWSON: Not in children.

13 DR. MARTY: As you well know, the data on
14 formaldehyde-induced exacerbation of asthma are mixed.
15 Some studies have said yes. Some studies have said no.

16 DR. BLANC: Okay. So I just want to make sure
17 that I understand what it is that you're arguing.
18 Because the implication, the one we just -- in the
19 earlier discussion could have been interpreted
20 differently, so I want to make sure that I understand
21 what it is that you're trying to say here.

22 So this is for -- and when we go down from our
23 generic arguments to the specific chemicals, this is an
24 example of a chemical which, based on its irritant
25 effects, the argument would be that -- in fact, the

1 argument here is not, in fact, anything to do with
2 asthma. It's just that the irritant effects of
3 formaldehyde you're arguing are greater in children than
4 they are in adults.

5 DR. MARTY: That's the main argument, yes.

6 DR. BLANC: And having nothing at all to do
7 with asthma at all. So it's not related to the argument
8 of how many children have asthma in the population?

9 DR. MARTY: Well, we used the potential since
10 I'm not completely convinced that asthmatics wouldn't
11 respond more than non-asthmatics to formaldehyde. We
12 use that as sort of another little piece of information.
13 But the real crux of the issue is this paper and the
14 impacts on measures of respiratory function being
15 greater in the kids in the study than in the adults. So
16 yes.

17 DR. FUCALORO: So in your main text when you
18 say "summary of potential for differential effects"
19 means there may not be differential effects because you
20 say "including cellular" and "exacerbation of asthma."

21 DR. MARTY: Right. That's right. Some of
22 those -- some of the effects we list have more weight
23 because the data are better and stronger. In the case
24 of formaldehyde --

25 DR. BLANC: The argument is that children are

1 more likely to have the irritant effects of
2 formaldehyde --

3 DR. MARTY: Yes.

4 DR. BLANC: -- at a dose more than for any
5 other irritant. Preferentially more, except for maybe
6 some other irritant that's in the list of 11. But, in
7 general, of all the irritants that one could look at,
8 formaldehyde is one at which -- given the levels of
9 ambient exposure, children would be more likely to have
10 an exaggerated irritant response --

11 DR. MARTY: Yes.

12 DR. BLANC: -- than adults even taking into
13 account their greater respiratory rate, et cetera, et
14 cetera, et cetera.

15 DR. MARTY: Yes. That's the crux of the --

16 DR. BLANC: And that's based on the
17 Krzyzanowski study.

18 DR. MARTY: Right.

19 DR. BLANC: I'm just trying to understand the
20 argument. Okay.

21 DR. DAWSON: And then I would just add that
22 these are quite low levels of concentrations we're
23 talking about.

24 DR. BLANC: I don't necessarily think that any
25 of the ancillary studies are that relevant to the

1 argument you're making since none of them are looking at
2 children versus adults, and the IGE argument is so far
3 off base because that's not the argument you're trying
4 to make. You're not trying to say that children are
5 preferentially sensitized to formaldehyde either because
6 the whole issue of sensitization is a big can of worms
7 that you probably don't want to get into.

8 You're certainly on much firmer ground when you
9 talk about irritant effects of formaldehydes than when
10 you talk about sensitization since even an occupational
11 population is included. It's exceedingly difficult to
12 demonstrate specific sensitization to formaldehyde which
13 makes the Vienna data seem very suspect since it's very
14 hard to show specific IGE reliably for formaldehyde.

15 DR. DAWSON: Well, just to respond to the one
16 key study in Vienna, again, I did look that up. The
17 rast values do drop when they move to the classroom. In
18 three months, the rast drops significantly.

19 DR. BLANC: Yeah. I understand. But what I'm
20 saying is it's difficult to understand what that rast is
21 because, technically, looking at a rast for
22 formaldehyde, it's a very, very -- it's one of those
23 murky, difficult, controversial areas is all I'm trying
24 to point out. There's a lot of pitfalls.

25 DR. DAWSON: And I hope I did mention that the

1 NO is -- the authors believed is a measure of
2 inflammation in the lower airways.

3 DR. BLANC: Right. That's just not a study
4 that has anything to do with whether the children have
5 more inflammation than adults. Nobody's arguing that
6 formaldehyde is not a pro-inflammatory irritant.

7 DR. MARTY: Okay.

8 DR. DAWSON: But, see, these are at low levels.
9 Very -- yeah.

10 DR. GLANTZ: Well, no one is arguing with that
11 either.

12 DR. BLANC: Yeah. That's not the point.

13 DR. MARTY: The other -- when you read the
14 document, we also do mention that it is a carcinogen.

15 DR. BLANC: Yeah. I know. I know.

16 DR. MARTY: It's a genotoxic carcinogen.
17 That's another reason to be worried about early
18 exposure.

19 DR. BLANC: Even though it's not exactly in
20 order, I think the chemicals are so related it would be
21 very interesting to hear, in light of your formaldehyde
22 presentation, your acrolein presentation, one juxtaposed
23 against the other.

24 DR. MARTY: We could do that.

25 CHAIRMAN FROINES: I don't think the

1 carcinogenesis argument any relevance, unless you're
2 prepared to state just what it is.

3 DR. MARTY: Only that there is concern among
4 lots of scientists that genotoxic carcinogens may be bad
5 actors if you're exposed early in life. That's the
6 concern. We didn't discuss it in the document other
7 than to mention it. We didn't want to get into this
8 argument over that specific issue since we are working
9 on that in a separate program and don't have all of the
10 information we'd like to have yet to develop that
11 argument.

12 MR. ALEXEEFF: It's just a little bit of an
13 aside -- George Alexeeff. We have a separate project
14 where we're developing guidelines for assessing
15 preferential carcinogenicity in children versus adults.
16 That's something we'll probably bring back. We'll
17 probably share it with this panel even though it's not
18 directly part of this project, but eventually it will be
19 part of it because it'll be part of the guidelines
20 ultimately on how we do those things.

21 CHAIRMAN FROINES: Well, I think that it will
22 be interesting. I think that short of an evidentiary
23 basis, there are -- one has to decide where are the
24 limits to speculation and a two sentence statement that
25 says "Genotoxic carcinogens may have relevance to kids,"

1 may be entirely correct, but that's reaching a pretty
2 high level of speculation with no evidentiary basis
3 associated with it. That's all my point is.

4 It's not to quarrel. But you might not be
5 correct. But it's hard for us to make a decision based
6 on something like that.

7 DR. MARTY: Should we go to acrolein?

8 CHAIRMAN FROINES: Sure.

9 DR. MARTY: Judy Polakoff is going to present
10 the information on acrolein.

11 MS. POLAKOFF: Okay. Acrolein was placed in
12 Tier 2 because data indicate that ambient concentrations
13 are above the chronic REL. Data suggests that acrolein
14 may exacerbate asthma. And exposure to various
15 pollutants, particularly reactive irritants, for
16 example, aldehydes, can increase bronchial
17 responsiveness to allergin stimulation or bronchial
18 reactivity.

19 DR. MARTY: I'd like to add that it ranked
20 first in our prioritization and had the highest ratio by
21 a long shot of --

22 CHAIRMAN FROINES: Can I make one comment?

23 DR. MARTY: Sure.

24 CHAIRMAN FROINES: I'd just like to say I think
25 that presentation is great. It's very succinct.

1 DR. ATKINSON: Okay. I have a question on the
2 ambient concentration.

3 DR. GLANTZ: But he hates it.

4 MS. POLAKOFF: Okay.

5 CHAIRMAN FROINES: We give and take it away.

6 DR. GLANTZ: Are you having fun yet?

7 MS. POLAKOFF: So much.

8 DR. ATKINSON: The ambient air concentrations
9 that you give for acrolein seem horrendously high --

10 MS. POLAKOFF: Well, we're going to get to
11 that. We'll get to that.

12 DR. ATKINSON: At least with respect to what's
13 being measured on -- or what was last measured in L.A.

14 MS. POLAKOFF: Okay. I'm going to get to that.

15 DR. ATKINSON: I mean, the data I've got from
16 what looked like the most recent comprehensive study in
17 L.A., which was -- admittedly it was 1993 data and
18 published in '96, but it had the -- a whole bunch of
19 carbonator compounds, and acrolein was an upper limit
20 that was a factor of 100 less than formaldehyde.

21 DR. FUCALORO: Less than formaldehyde?

22 DR. ATKINSON: Much less. Yeah. Unless you're
23 sitting somewhere, I assume, by a place which is
24 emitting acrolein, a direct emission place, and not a
25 vehicle, I don't see how it could be higher than

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1 formaldehyde. I mean, if you look vehicle exhaust, both
2 diesel and light duty, acrolein is significantly less
3 than formaldehyde as an emission.

4 DR. MARTY: We have some information on other
5 pieces of data that we found on acrolein measurement and
6 model concentrations.

7 DR. ATKINSON: Yeah.

8 CHAIRMAN FROINES: Wait a second. Taking the
9 prerogative of the chair, why don't we give her a chance
10 to present some data, then Roger can quarrel with it,
11 but let's have her give her statement and then --

12 DR. ATKINSON: Okay.

13 DR. MARTY: Andy, could you go back?

14 CHAIRMAN FROINES: Who published -- who's work
15 is that?

16 DR. ATKINSON: This is Grandjean. It was
17 published in '96. This is the last recent one with a
18 whole bunch with acrolein.

19 I mean, your document actually says there's
20 little data on acrolein.

21 DR. MARTY: Yeah. Andy, can we have the next
22 slide. Sorry.

23 DR. ATKINSON: Sorry.

24 MS. POLAKOFF: Let me start by saying that
25 acrolein is extremely difficult to measure. The Air

1 Resources Board has very little exposure data for us on
2 acrolein, and the staff that we've spoken to have, you
3 know, indicated that they don't have a lot of confidence
4 in many of the measurements that are out there because
5 it is so difficult to measure.

6 Now, having said that, these are the values
7 that we found in the literature.

8 Andy, if you could do the next slide.

9 Here's also concentrations from cigarette
10 smoke.

11 DR. MARTY: Can I add something there, too?
12 When talking with ARB with Mike Pore, his concern about
13 their measurements of acrolein were that they were
14 underestimating because of the reactivity of acrolein
15 and the methods they were using for sampling.

16 DR. ATKINSON: Okay.

17 CHAIRMAN FROINES: Could you go back to that
18 just for a second? So that the California data are the
19 top three?

20 MS. POLAKOFF: Yeah. The first two studies,
21 they're very small. The first two are really small.
22 The first one was 13 outdoor measurements. CARB took
23 that data from Woodland California, and many of the
24 measurements, I think, were below the level of
25 detection, so that it's just a few numbers there. It's

1 not -- they're not really confident in those data I have
2 to say.

3 CHAIRMAN FROINES: What does this paper say,
4 Roger?

5 DR. ATKINSON: The paper I've got says that
6 acrolein was observed in every measurement. There was
7 32 measurements, but in each case it was very close to
8 their detection limit, and they put a number of less or
9 equal to 0.04 ppb. And that was a --

10 DR. FUCALORO: 0.04?

11 DR. ATKINSON: Yeah. And formaldehyde was 5.3.
12 There's an average of 32 measurements, and I think they
13 were taken at four stations in L.A., Long Beach, Azusa,
14 Claremont was one. I can't remember the other one.

15 DR. GLANTZ: What does that convert to in
16 micrograms.

17 DR. ATKINSON: You multiply by roughly a factor
18 of two, so it's about .1. Less or equal to is the way
19 it was written in the paper.

20 DR. FUCALORO: So a hundred times different --
21 a thousand times different.

22 DR. ATKINSON: A hundred.

23 DR. BLANC: But, in all fairness, you have the
24 California Air Resources Board data that they're
25 presenting to us disagrees with that and is within the

1 range of U.S. EPA estimate, roughly.

2 DR. ATKINSON: Yeah. I mean, don't forget

3 those data are not exactly -- apart from the second

4 California one, the first one is 1990 data. And I

5 suspect that the numbers are going down. At least

6 emissions from vehicles are probably going down.

7 DR. FUCALORO: Well, isn't the 14.3, that is

8 the U.S. -- I am sorry.

9 DR. ATKINSON: I don't know.

10 DR. FUCALORO: It's U.S. EPA data, at least by

11 looking at that slide.

12 DR. FUCALORO: To 1980.

13 DR. ATKINSON: 1961 to 1980, yeah.

14 DR. BLANC: Well, there's a lot of exposure out

15 there anyway, in your view?

16 MS. POLAKOFF: Yes.

17 DR. MARTY: Yes.

18 CHAIRMAN FROINES: Both as a primary

19 pollutant -- in terms of primary emissions but also as

20 an atmospheric chemistry product from butadiene?

21 DR. ATKINSON: Well, that's the only thing that

22 forms it. I mean, formaldehyde is formed from every

23 VOC, essentially.

24 MS. POLAKOFF: One more, Andy. Thanks.

25 U.S. EPA did extensive modeling work as part of

1 their cumulative exposure project, and they have
2 modeling data for 148 hazardous air pollutants,
3 including acrolein. And from that data, it's estimated
4 that the annual, average ambient concentration of
5 acrolein in California is 0.15 micrograms per cubic
6 meter.

7 DR. GLANTZ: That's about what Roger said.

8 DR. FUCALORO: Yeah.

9 MS. POLAKOFF: Okay. Pratt et al., did a study
10 examining the SEP data, the 1990 data, and did a study
11 for Minnesota using the model data and some monitoring
12 data looking at air toxics in Minnesota. They used a
13 hazard quotient approach and compared exposure data.
14 They used the modeling data from U.S. EPA, as well as
15 monitoring data where they had it, and they compared
16 that exposure data to cancer and non-cancer health
17 benchmark values.

18 They only had modeling data for acrolein. They
19 looked at over 1,200 census tracts in Minnesota and
20 found out that for 70 percent of the census tracts
21 studied, 70 percent of the census tracts exceeded the
22 benchmark for acrolein.

23 Next slide. They also estimated a screening
24 level total hazard index by summing all of the
25 non-cancer hazard quotients over all endpoints. And

1 acrolein was by far the most important contributor to
2 the non-cancer hazard index. Eighty-nine percent of the
3 risk was attributed to acrolein. The next highest
4 chemical was formaldehyde at 6 percent, and each of the
5 other pollutants accounted for less than 1 percent.

6 And where they could compare their modeling
7 data with the monitoring data, they found that the
8 tendency was to under-predict measure values, which is
9 what Melanie had mentioned before from ARB.

10 Next slide. Although there is no direct
11 evidence of a link between acrolein exposure and asthma,
12 the data do suggest that acrolein may exacerbate asthma
13 in humans.

14 Next slide. This study was conducted in guinea
15 pigs. The authors were looking at leukotrienes and
16 acrolein-induced bronchial hyper responsiveness. The
17 reason that they're looking at leukotrienes is that in
18 airways, leukotrienes active mucous secretion and smooth
19 muscle contraction and are thought to be important in
20 the pathophysiology of asthma. So they wanted to see if
21 they blocked leukotriene receptors with an antagonist or
22 if they blocked the synthesis of leukotrienes, whether
23 this would diminish the acrolein-induced
24 broncho-responsiveness in guinea pigs.

25 And they also wanted to measure concentrations

1 of a specific leukotriene, the LTC 4, to see if it
2 was -- if concentrations increased in lavage fluid if
3 they found an increase in falling acrolein exposure.

4 Okay. And this slide along the Y axis a
5 specific pulmonary resistance, and along the X axis is
6 time. These two graphs could actually be superimposed
7 on each other. On the Y axis, the scales are the same.
8 They're just separated out for clarity I think. That's
9 how the authors did it.

10 So in this part of the experiment, guinea pigs
11 were exposed to 1.3 part per million acrolein for two
12 hours, and then the graph shows broncho-constriction
13 immediately following acrolein exposure. The top bar --
14 the top line with the open circles is just acrolein.
15 And so acrolein alone, you see, increases specific area
16 resistance, and this effect lasts about an hour.

17 Underneath it is the effect if the animals are
18 given either the leukotriene receptor antagonist or the
19 synthesis inhibitors prior to acrolein exposure, the
20 effect is diminished, or at least it's delayed in some
21 of the cases.

22 DR. BYUS: It goes up in the control, too,
23 doesn't it? Did they give the control?

24 MS. POLAKOFF: The control is really just
25 the --

1 DR. BYUS: No inhibitors?

2 MS. POLAKOFF: No inhibitors. Right. It's

3 just acrolein by itself which is the top one.

4 DR. BYUS: No. I mean the lower panel, the

5 control, did that get the inhibitors?

6 MS. POLAKOFF: The lower two -- the lower two

7 are with synthesis inhibitors. Correct.

8 DR. BYUS: Both of them; right?

9 MS. POLAKOFF: Yeah. Different inhibitors.

10 DR. BYUS: It went up?

11 MS. POLAKOFF: One of them kind of delayed, and

12 one of them diminished.

13 DR. BYUS: But the control in the lower panel

14 went up when they gave the inhibitor without acrolein.

15 MS. POLAKOFF: They don't have it without --

16 all the animals are given acrolein. It's just whether

17 or not they're given it before the acrolein exposure.

18 DR. BYUS: I'm just saying the lower -- in

19 Panel B, the control, which I assume is the solid -- is

20 the triangles, solid triangles, and was given inhibitor,

21 two, leukotriene synthesis, that also increased airway

22 resistance.

23 DR. MARTY: Actually, those animals were given

24 acrolein after being given the inhibitor. So all four

25 of those lines, the animals were being exposed to

1 acrolein.

2 DR. BYUS: Oh, all right. Okay.

3 MS. POLAKOFF: Sorry.

4 DR. BYUS: Sorry. No problem. It's what you

5 call the controls that's always confusing. We always

6 call them something different.

7 DR. MARTY: The control is actually the

8 treated.

9 DR. BYUS: Okay. Okay. Okay.

10 MS. POLAKOFF: Okay. In this slide, the Y axis

11 is the effective dose 200 or the concentration of

12 acetylcholine that causes a doubling of the specific

13 airway resistance. So this is the dose that's needed to

14 get the response. So the open bars are just

15 acetylcholine, so that's providing the baseline.

16 After that, the animals are exposed to 1.3 part

17 per million acrolein for two hours, and after the

18 acrolein exposure, then they're given acetylcholine one

19 hour, two hours, six hours for twenty-four hours after

20 the acrolein exposure.

21 So following the acrolein exposure, it takes

22 much less acetylcholine to cause the same doubling of

23 airway resistance. So, therefore, acrolein appears to

24 sensitize the lungs to hyper-respond, and this effect is

25 seen even at 24 hours.

1 Okay. Now, this graph -- or these graphs are
2 showing what happens when the animals are given either
3 the leukotriene synthesis inhibitors or the leukotriene
4 receptor antagonist just prior to acrolein exposure.
5 The upper and lower graphs are where the leukotriene
6 synthesis inhibitor was given. The middle one is
7 showing the leukotriene receptor antagonist.

8 So starting from the left, the open bars are
9 the control or the baseline. The animals are just given
10 acetylcholine. The hatch bars, PD, is post-drug. So
11 that's showing given just the leukotriene receptor
12 antagonist or the synthesis inhibitor, there's no effect
13 on the effective dose, the ED 200.

14 Then acrolein is given to all the animals.
15 acrolein exposure 1.3 part per million for two hours,
16 and then following the exposure, again it's the
17 acetylcholine one hour, two hours, six hours or
18 twenty-four hours after. And it's certainly not the
19 picture we saw on the slide before without the
20 inhibitors or the antagonist.

21 So, basically, to kind of summarize the
22 results, acrolein exposure produced this transient
23 increase in pulmonary resistance that was reversible
24 after the cessation of exposure. It lasted about an
25 hour.

1 Acrolein decreased the effective dose of ED 200
2 of acetylcholine necessary to double specific airway
3 pulmonary resistance in exposed animals, and that effect
4 lasted about 24 hours. The leukotriene receptor
5 antagonist and the leukotriene synthesis inhibitors
6 attenuated the acrolein-induced hyper-responsiveness.

7 And then the last part of that experiment, the
8 authors measured concentrations of a specific
9 leukotriene, the LTC 4, and they found that it did
10 increase in the broncho-alveolar lavage fluid after
11 acrolein exposure. And when they gave the synthesis
12 inhibitors, they did not see that increase in that
13 leukotriene.

14 In addition to the broncho-reactivity, acrolein
15 causes mucous hypersecretion. In rats, tracheal mucin,
16 messenger RNA and mucin glycoproteins were elevated in
17 lung tissues following in vivo exposures to 3 part per
18 million acrolein, six hours a day for two weeks.

19 Similarly in mice, acrolein exposure resulted
20 in significant increases -- in this case, macrophages
21 and neutrophils they found in the fluid, which are
22 indicative of the inflammatory response, along with the
23 increased mucin, messenger RNA synthesis and secretion.

24 The next slide. Human invitro data, results
25 from two studies are summarized here. The first is also

1 by Borchers who is from the previous slide. It was
2 reported that invitro acrolein can act directly on
3 airway epithelial cells to increase mucin messenger RNA
4 levels.

5 In the second study, this is from a different
6 laboratory, Ru et al. 1999. These investigators were
7 looking at the interaction between passive sensitization
8 of human isolated airways and acrolein exposure. They
9 took lung tissue from non-atopic, non-asthmatic
10 patients, and they bathed the tissue in the sera from
11 atopic asthmatic patients, and they reported that the
12 passive sensitization, in addition to acrolein exposure,
13 have a combined effect on the bronchial smooth muscle
14 reactivity in response to different agonists.

15 In the tissues that were sensitized by
16 incubation, pre-exposure to acrolein for either 10 or 20
17 minutes, resulted in a significant increase in the
18 maximum contractile response to either a specific or
19 non-specific agonist.

20 And so, just to summarize, we don't have
21 evidence of a direct effect. We have a large number of
22 studies that indicate that allergic airway diseases,
23 including asthma, are associated with air pollution, of
24 which acrolein is a component.

25 The Leikauf study, which was the first study,

1 described studies in the guinea pig of acrolein-induced
2 hyper-responsiveness to acetylcholine and
3 broncho-constriction, which could be considered analogs
4 of response in asthmatic humans exposed to reactive
5 irritants.

6 Clinical studies, as well as animal studies,
7 have shown that exposure to various air pollutants,
8 particularly reactive irritants, can increase
9 responsiveness to allergens in relation to
10 broncho-reactivity. And formaldehyde is a better
11 studied example of that.

12 Studied invitro acrolein potentiated the
13 contractile response of immunologically sensitized human
14 bronchial tissue to specific antigen stimulation.

15 In animals, acrolein exposure causes mucous
16 hypersecretion. And in isolated human cells, acrolein
17 increased mucin messenger RNA levels.

18 CHAIRMAN FROINES: Thank you. We're going to
19 take a break shortly, but why don't we have some
20 discussion before we take a break?

21 DR. ATKINSON: So based on what I've seen of
22 the ambient data in L.A., I would suggest that the
23 chronic REL -- or the air concentration divided by the
24 chronic REL for acrolein and formaldehyde are probably
25 pretty similar.

1 DR. FUCALORO: Would be what?

2 DR. ATKINSON: Similar.

3 DR. MARTY: Similar. It's true. It is --

4 DR. ATKINSON: I don't dispute that acrolein --

5 if you take the air concentration divided by the REL,

6 acrolein may indeed be higher than formaldehyde, but I

7 would be surprised if it's 200 times.

8 DR. MARTY: Good point.

9 DR. FUCALORO: If you reduced the concentration

10 by a factor of 100 as you had previously suggested --

11 DR. ATKINSON: No. That comes down to two to

12 one. Yeah.

13 DR. FUCALORO: Yeah.

14 DR. BYUS: So could you just -- the child

15 sensitivity issue now, I mean it's -- could you just --

16 where are we?

17 DR. MARTY: What's the connection?

18 DR. BYUS: What's the connection? Yes.

19 DR. MARTY: Okay.

20 DR. BYUS: Is it just that children are more

21 likely to have asthma?

22 DR. MARTY: The connection is -- exactly. The

23 discussion we had earlier where we are viewing asthma as

24 the disease that impacts children disproportionately.

25 DR. BYUS: Okay.

1 DR. MARTY: And we had evidence here on a
2 biochemical level and in vivo animal studies showing
3 that acrolein is capable of doing of what asthmatic
4 exacerbants can do: Hyper-responsiveness of the airway,
5 increase the mucin secretion.

6 DR. BYUS: But there's no direct evidence that
7 it does that any more or less or the equivalent in
8 children?

9 CHAIRMAN FROINES: You won't find any human
10 data. It's because acrolein is so much part of air
11 pollution that you won't find any, you know, unique
12 exposures in
13 a --

14 MS. POLAKOFF: Well, it's too hard to measure.

15 CHAIRMAN FROINES: I just -- not that I'm aware
16 of.

17 DR. BLANC: So comparing head on to
18 formaldehyde and acrolein, acrolein is a more potent
19 irritant. Based on your data, the ratio, the exposures
20 to REL is certainly much higher for formaldehyde and
21 you're discounting --

22 CHAIRMAN FROINES: Acrolein.

23 DR. BLANC: And you're discounting acrolein and
24 even discounting your air levels somewhat, which,
25 perhaps, you shouldn't discount because you have data

1 from the Air Resources Board saying they believe they've
2 underestimated, you would still come out higher than
3 formaldehyde even if you significantly discount it.

4 So I think the truth has to be somewhere
5 between the data you have and the data they have because
6 that's -- because we know that we're underestimating.
7 We don't believe that that's the same problem as with
8 formaldehyde. The only thing that --

9 DR. BYUS: The biochemical thing is much better
10 for acrolein. Much better.

11 DR. BLANC: Well, the data we were presented.

12 DR. BYUS: Yes. The one we were presented.

13 DR. BLANC: There is a lot of literature out
14 there on formaldehyde, but it's certainly been better
15 studied in controlled human exposures. But we know that
16 acrolein is much more potent than formaldehyde and is,
17 generally speaking, under-regulated relative to
18 formaldehyde I would say.

19 So the only thing that's driving you is the
20 Krzyzanowski study, not of asthmatics, but where the
21 peak flow in children -- where they didn't measure
22 acrolein and there probably was co-exposure with
23 acrolein, and the two tend to run parallel also in the
24 kinds of environments they were looking at probably,
25 that you would favor acrolein were it not for your

1 interpretation of the Krzyzanowski study; is that a fair
2 characterization?

3 DR. MARTY: Yes. I would add a little bit to
4 that. We were unsure enough about the concentrations in
5 air that even though acrolein scored way high, we were a
6 little bit reluctant to put it in Tier 1. That may have
7 been not a good decision. I don't know.

8 We also were concerned about the ratio of the
9 ambient data to the REL. And even if you divided by
10 100, you're still above the REL, and you have about the
11 same ratio of formaldehyde and acrolein.

12 CHAIRMAN FROINES: Can I ask you a question
13 that goes to Paul's? In terms of -- the guinea pig data
14 is -- it's a nice, solid set of data, and so it's
15 compelling because it's clear and direct, and you can
16 live with it and --

17 DR. MARTY: And if you're a toxicologist, you
18 like that.

19 CHAIRMAN FROINES: Toxicologists love that.
20 That's exactly why we do toxicology.

21 What's the comparable literature? Because
22 since you don't point out any animal literature on
23 formaldehyde, does that mean that the data is by and
24 large negative? Does that mean that there's not data
25 that you think is relevant or what -- clearly people

1 have been studying formaldehyde much more than acrolein.
2 So what is the circumstances? What are the
3 circumstances?

4 DR. BLANC: Depends on who they delegated
5 the --

6 DR. MARTY: That's a good question.

7 DR. BLANC: -- the literature review to,
8 doesn't it?

9 DR. MARTY: Well, we --

10 DR. BLANC: I doubt the literature review was
11 done by the same person, was it?

12 DR. MARTY: No, they were not. I think what we
13 did with formaldehyde, because we had so many studies,
14 actually, in people, that we did emphasize those. But
15 we can go back and look at to see if there are any of
16 the same sorts of data at the biochemical level for
17 formaldehyde as there are for acrolein.

18 My guess is probably not because -- because of
19 this issue of people saying, "Well, we don't think it
20 really exacerbates asthma," unless you've had
21 occupationally-induced formaldehyde-specific asthma.

22 So I don't know if that data are there. They
23 certainly didn't pop up in the search that was done.

24 DR. ATKINSON: And the other thing you have to
25 be careful about is comparing ambient data from one

1 decade to a decade differently because the
2 concentrations have been decreasing quite steadily. If
3 you look at formaldehyde in the L.A. Basin, they've gone
4 down by a factor of about ten in the last 20 years or
5 so. Twenty to thirty years.

6 And it seemed every time they do a field study
7 and do extensive measurements, the concentrations are
8 lower than previously.

9 DR. FUCALORO: Roger, can you help me on this?
10 Just looking at the formula for acrolein, it looks like
11 it's a type of product that wouldn't last long in the
12 environment. It seems to be pretty --

13 DR. ATKINSON: It's pretty -- yeah. But
14 formaldehyde has an even shorter lifetime. Formaldehyde
15 photolyses -- well, acrolein my photolyze. We don't
16 know enough about its lifetime.

17 I mean, the other one is that acrolein can only
18 be formed in the atmosphere from dyeing, such as
19 1, 3-Butadiene, whereas formaldehyde is formed from
20 almost all organics. In L.A. it's believed that
21 something like 80 percent of the formaldehyde is formed
22 in the atmosphere.

23 DR. BLANC: I guess my bottom line would be
24 from where I sit with the information that you've given
25 in my role, you know, as a scientific, tertiary

1 reviewer, that I think the argument is more compelling
2 for acrolein to be in the top five than for formaldehyde
3 to be in the top five.

4 I guess I wouldn't -- you know, I'm not going
5 to get in the argument about whether or not formaldehyde
6 should have made it from your list of 35 into some
7 shorter list. I don't think it's reasonable given all
8 the questions that you have to have done that step. But
9 I think that prior to your next submission of a revised
10 document, you should think very long and hard about the
11 relative position of those two chemicals.

12 Now, I think you have a problem in sort of
13 weighting -- given the nature of the exercise that
14 you're going through and the regulatory implications,
15 you're probably -- you've probably made the right choice
16 by not including both aldehydes in the same short list
17 because it would really be sort of really dominating
18 what was driving the five chemicals.

19 So I think your inclination to choose between
20 the two of them was probably appropriate in taking the
21 global challenge of what you were trying to do. But my
22 own inclination, based on the information you've
23 provided so far, would be that the evidence weighs in
24 favor of acrolein in a relative basis. And that would
25 be driven, I think, by its -- the potency of its

1 irritancy, the scenarios for exposure, including from
2 combustion products and indoor sources, and its relative
3 under-attention from a regulatory point of view.

4 And one of the goals of the legislation was to
5 make -- to force the Air Resources Board to take a hard
6 look at a short list of chemicals in ways that could
7 drive control steps. Then this would be one of the ones
8 I would say, "Yeah. Take a hard look at this one."

9 CHAIRMAN FROINES: I want to make one comment
10 to Tony. The one thing that's interesting from a
11 toxicologic standpoint, chemical structure standpoint of
12 acrolein, is acrolein is, you know, a double bond
13 connected to an aldehyde group, and so that compound
14 undergoes mycliditions (phonetic) with nucleophiles, so
15 it is a very powerful electrophile in that respect.

16 DR. FUCALORO: That's why I said I didn't
17 expect it to last long in the environment.

18 CHAIRMAN FROINES: Right. And so therefore,
19 without getting into -- the problem is people have
20 studied the carcinogenicity of formaldehyde pretty
21 extensively. There is a database there. People have
22 not studied the carcinogenicity of acrolein to the
23 degree that one would like. But I would suggest that
24 acrolein is likely to be a carcinogen, and I think over
25 time we'll find that proves out to be the case.

1 So I tend to agree with Paul in terms of his
2 conclusion because the compound -- although, Roger is
3 right insofar as there are widespread sources of
4 formaldehyde, as we know, and acrolein is more limited
5 in that respect. But toxicologically, I think the
6 argument might favor acrolein. So it's a close call in
7 any way, in any circumstance.

8 DR. FUCALORO: Of course, there's no unit risk
9 factor given for acrolein. But you said that's because
10 of the --

11 CHAIRMAN FROINES: Yeah. It's the vacuum, not
12 the negative data. And I think it's worth considering
13 Paul's argument about if we're trying to get ARB's
14 attention with respect to approaching some of these
15 things that haven't gotten attention, then acrolein is a
16 very good candidate for that.

17 Why don't we take a five- to ten-minute break,
18 and then we'll -- sorry, Melanie.

19 DR. MARTY: Can I just make one quick comment?
20 There are data looking at formaldehyde and already 50
21 studies, for example, and in guinea pig models of hyper-
22 responsiveness. It may be worthwhile to flesh that out
23 more in the document and bring it to the panel.

24 CHAIRMAN FROINES: Well, in this case, I think
25 it's important to try and -- since we're obviously

1 probably going to argue in favor of one versus the
2 other, the way -- at least the way the discussion has
3 gone, it's good to have some sort of comparability in
4 the information we have to work with.

5 DR. ATKINSON: I mean, the funny one is that
6 the same database has crotonaldehyde, which is the next
7 log up, ten times higher than acrolein.

8 CHAIRMAN FROINES: See, if I express my bias,
9 it would be that we have PAHs; right? Nobody worries a
10 bit about PAHs. I would argue that we should have
11 aldehydes and have acetaldehyde, formaldehyde, crotonaldehyde,
12 glutaraldehyde, acrolein and probably a couple
13 others, and it would make perfect sense, but we probably
14 won't do that. But if you're arguing by analogy, we
15 should.

16 DR. MARTY: If it makes you feel better, I
17 think in terms of engineering controls on combustion
18 sources to reduce one aldehyde -- and Roger can correct
19 me if my assumption is wrong -- you would be reducing
20 most of the aldehydes.

21 DR. ATKINSON: Yeah, yeah.

22 CHAIRMAN FROINES: And that is the precise
23 argument that a former ARB staff person made when I
24 complained about doing benzopyrene years ago. She said,
25 "If we do benzopyrene, we'll control all the PAHs." And

1 what was the comment you made earlier about which
2 compounds have not had control strategies developed?
3 PAHs. So that the notion of doing benzopyrene and PAHs
4 hasn't driven the process, so that obviously we need a
5 different hook. Thanks.

6 We'll take a break.

7 (Recess.)

8 CHAIRMAN FROINES: We have this room until
9 5:00, so we're going to --

10 DR. GLANTZ: Talk really fast.

11 CHAIRMAN FROINES: -- talk really fast and
12 cover all eight of the rest of the compounds. And
13 the -- I think -- is Jim Behrmann here someplace? My
14 guess is that we're going to finish going through these
15 compounds at the May 14th meeting so that I think that's
16 the next phase of this.

17 In talking with Melanie and George at the
18 break, we talked about what are people's energy levels
19 up to, and I think that we talked about doing lead and,
20 perhaps, mercury in the next hour because, presumably,
21 they are enormous amounts of data, but they're
22 relatively straightforward at some levels as well.

23 DR. GLANTZ: Can I just ask one question?

24 CHAIRMAN FROINES: Sure.

25 DR. GLANTZ: I have to get Melanie's attention.

1 Melanie. Yoo-hoo.

2 DR. MARTY: I'm sorry.

3 DR. GLANTZ: It's okay. When we come back on

4 May 14th, are you going to have done anything to this

5 document or proposed shuffling lists around or any of

6 the -- we had our extremely long discussion this morning

7 about, you know, why -- coming up with sort of why you

8 did what you did and all of that. Are you going to have

9 any of that for us to look at by the next meeting? It

10 would be nice.

11 DR. MARTY: We'll try to have some of them.

12 We'll try to have the things that you asked us to do in

13 the introduction done.

14 DR. GLANTZ: Okay.

15 DR. MARTY: In terms of adding either

16 additional summaries that -- for example, for chemicals

17 that Paul mentioned that are important --

18 DR. GLANTZ: Yeah.

19 DR. MARTY: I'm not sure that we can have that

20 done. I realize that gives us one week to do things

21 because you folks need to get the document with some

22 time to look at it.

23 DR. GLANTZ: Okay. The one thing -- I think

24 that would be very helpful. And, I mean, one other --

25 if, as a result of the discussions today you wanted to

1 propose shuffling things around on any of these lists, I
2 think if you were to do that before then, I would --
3 feel free to do it, you know. If not, that's okay too.

4 But, you know, just in the interest of -- well,
5 no. In the interest of moving things along. I think
6 that there have been -- as a result of the discussion
7 this morning and some of the things that were said
8 today, you know, you might want to come back to us with
9 some changes in the priorities, and the sooner we get to
10 see those, the better I think. If not, we'll probably
11 get to meet a couple more times about this before July.

12 CHAIRMAN FROINES: My guess is that to ask them
13 to do much changing and improving of the document is
14 probably not feasible given they have a week but -- so I
15 would focus on trying to make, you know, the best
16 presentation of the remaining chemicals so that the
17 issues are as succinct as possible to help facilitate
18 the process rather than trying to --

19 DR. GLANTZ: Okay.

20 CHAIRMAN FROINES: Scurry around and writing,
21 doing a --

22 DR. GLANTZ: Yeah. That's probably true. But
23 I think, like, one of the things, though, from this
24 morning was the idea that the Tier 2 might get to be a
25 bit longer list, so I think if that were the case, it

1 would be nice to at least get presentations on the
2 things that you thought ought to be on the -- any
3 additional compounds on the Tier 2 list. You know,
4 based on what was discussed this morning. We might
5 not, you know?

6 CHAIRMAN FROINES: My guess is --

7 DR. GLANTZ: Well, why don't you go on?

8 CHAIRMAN FROINES: -- that we'll be -- there
9 will be 11. We need to get through this by the end of
10 May 14th. Not necessarily make every decision by May
11 14th, but hopefully make our decisions by May 14th.

12 DR. GLANTZ: Well, that's true. But that's why
13 I suggest that if the result is that some compounds are
14 going to be added into the Tier 2 list based on the
15 discussion this morning, that we should have some kind
16 of presentations about that.

17 CHAIRMAN FROINES: I have one question. Is
18 George -- there's George. Sort of a policy level
19 question. George, let's go -- let's assume that May
20 14th we go through -- get through all the 11 compounds,
21 and the panel continues to have suggestions about
22 changes in the document. The first question, I guess,
23 is when do you need to have a document that goes to ARB
24 for its consideration on July 1st?

25 And the second question is, Can you go into --

1 can you give the ARB a list of the five and take some
2 time to develop the document so that the underpinnings
3 for the decision actually doesn't necessarily get there
4 by July 1st, but you can get them a more complete
5 document, say, August 1st or something? I mean, in
6 other words, I'm trying to figure out because there's --
7 obviously, we're under a very tight time constraint, and
8 the question I'm really asking is, How are we going to
9 deal with the constraints?

10 DR. ALEXEEFF: Well, the way the statute reads
11 is it's actually the OEHHA director that has to make the
12 decision by the end of June -- I think it's by July 1st
13 he has to identify the top five chemicals.

14 Okay. Now, we have to do that in consultation
15 with the Air Resources Board. Now, we've already been
16 consulting with the Air Resources Board. So the Air
17 Resources Board does not have to make a decision in this
18 process. We're planning, once we're done with this, to
19 make a presentation to the Air Resources Board because
20 then they have to look at their responsibilities under
21 the act.

22 So -- but we -- we thought it would be great if
23 we could have wrapped the whole thing up by July 1st,
24 but based upon the issues that you raised I don't --
25 wrap the whole thing up meaning make a presentation to

1 the Air Resources Board as well, but that was our
2 original intention.

3 It doesn't look like it's going to happen based
4 upon the timing. But it's not required to happen by
5 law. What's required to happen is we have to come up
6 with the list of five by July 1st.

7 CHAIRMAN FROINES: And the -- and the
8 supporting document there's no time restriction.

9 MR. ALEXEEFF: I don't think there's a
10 requirement for a supporting document, but the basis has
11 to be reviewed -- let me just pull that statute up. The
12 basis has to be reviewed by the Scientific Review Panel
13 and then -- okay. So by July 1st of this year, "The
14 office, in consultation with the State Board, shall
15 establish a list of up to five TACs"; okay? "that may
16 cause infants and children to be especially susceptible
17 to illness." So that's by July 1st.

18 Okay. Then it says, "The office shall submit a
19 report containing the list and its reasons for including
20 the toxic air contaminants on the list to the SRP." And
21 then the SRP -- so we, quote, have "done that"; right?
22 Then it says, "The SRP, in a manner consistent with" the
23 other stuff that you do "shall review the list of TACs
24 submitted by the office, and as part of the review, any
25 person can submit other information to the panel." You

1 know, public comment type of period. So that's
2 basically the way the process is laid out.

3 So I think that the basis for the five should
4 be all crystal clear, if there's five, by July 1st and
5 that we have to list them by July 1st. Whether or not
6 the report is published and finalized is probably not
7 supercritical, but the closer it is, I think -- I think
8 we would probably plan on doing it by -- have it all
9 done by July 1st. That would be our -- we would
10 probably move everything -- all the mountains we could
11 to get it done by then.

12 DR. MARTY: We have to because I'm going on
13 vacation July 2nd.

14 DR. FUCALORO: You were going on vacation.

15 DR. GLANTZ: And she's never coming back.

16 CHAIRMAN FROINES: Let's go ahead. I think
17 that's clear. I think it puts a lot of -- it will put a
18 lot of emphasis on our really moving the process along
19 on May 14th so we bring it to closure from our
20 standpoint, because we'll want to write some level of
21 findings for ourselves as well.

22 DR. MARTY: There's another meeting planned in
23 June, yes? Peter.

24 MR. ALEXEEFF: It would probably be useful to
25 have a meeting planned in June.

1 CHAIRMAN FROINES: That's fine. This panel
2 decided to have a meeting every two months not long ago.
3 And, of course, we follow it up by planning three
4 meetings in two months. So we're doing very well.

5 DR. FUCALORO: Why don't we all get jobs at the
6 same university.

7 DR. COLLINS: You're the dean.

8 DR. FUCALORO: Former dean.

9 DR. GLANTZ: Well, most of us do have jobs at
10 the same university.

11 CHAIRMAN FROINES: Well, I would be quite happy
12 if the governor gave us a bunch of FTEs at you UCLA and
13 we had everybody move to Los Angeles. I'm not sure
14 Roger and Craig and Stan would buy into it, though.
15 I've been trying to get Paul to do it for years.

16 DR. BLANC: We should start. Really we need to
17 start.

18 CHAIRMAN FROINES: Melanie.

19 DR. MARTY: The next chemical we're going to
20 talk about is lead, and I just want to preface it by
21 saying this panel has looked at lead as a TAC not all
22 that long ago. The information focused on developmental
23 neurotoxicity and effects in children. We didn't think
24 we needed to review in detail that information again
25 today, so we have a pretty brief presentation.

1 DR. WINDER: So lead was selected for Tier 1
2 for these reasons: It's well documented to have very
3 extensive neurotoxic and developmental effects. And
4 these neurotoxic effects are more pronounced during
5 exposure in early childhood. There is some evidence for
6 prenatal exposures as well.

7 And low level exposures, in this case the
8 literature talks about 20 or 30 micrograms per deciliter
9 in the blood, are associated with developmental delays,
10 decrements in intelligence, memory, visual motor
11 function, perception integration and behavior.

12 Now, no known data so far support a distinct
13 threshold for effect. And the other reason for
14 considering lead in child-specific behaviors seem to
15 be -- involve making kids more at risk. Also, just
16 child-specific physiology, for example, the absorption
17 of lead is much more rapid in kids two years and
18 younger.

19 So we're summarizing, as Melanie was saying,
20 the major studies that were involved in this. Up here
21 we show the coefficients which were associated with the
22 effects of lead on intelligence using the WISC-R
23 intelligence scale, the revised version. And these are
24 broken down both into crude models and adjusted models,
25 as well as meat-analyses.

1 In all cases, we've shown the coefficients of
2 the correlation here in the right-hand column, and, in
3 general, you'll see a familiar correlation between the
4 levels in blood and performance levels on these
5 intelligence tests, and this seems to hold throughout
6 all these analyses.

7 Then this next table, this is something that
8 OEHHA worked up to try and examine what would happen at
9 certain blood lead levels. Now, we show on the left-
10 hand column our average air lead concentrations in
11 micrograms per cubic liter. The top one being the --
12 roughly the current level in California for the ambient
13 lead.

14 And then the next column over where it shows
15 the geometric mean of 3.14, this is from NHANES. This
16 is the, at that time, average across the United States.
17 And then each of the subsequent columns are kind of what
18 if kind of situations. If we reduce the lead in the
19 blood or if we change the geometric standard deviation,
20 how many children does this push above that magic number
21 of 10 micrograms per deciliter of blood lead? And so as
22 you see here, with the -- in a minor decrease in the
23 geometric mean where you see substantial decreases in
24 the percentage of children which will actually end up in
25 that above 10 micrograms per deciliter.

1 And as we approach the bottom of the graph
2 here, the 1.5 micrograms per cubic meter, our current
3 regulatory level, as much as 45 -- 46 percent of the
4 kids will move into this above 10 microgram per
5 deciliter level.

6 CHAIRMAN FROINES: I just have one question.
7 Is the -- what is -- you have two GSDs say at the top of
8 the row, and what's the basis for those? Are they from
9 NHANES calculations? Are they -- one is for one year
10 olds and the other is for two year olds? I don't quite
11 understand that table.

12 DR. MARTY: Yes, that's exactly right. The --
13 the geometric mean of 2.1 represents kids who are ages 1
14 and 2. And I think the other geometric mean is older
15 kids.

16 DR. FUCALORO: And the number you cited, the
17 .055, the document says that the California 1999 was
18 actually lower than that, 0.014, according to the
19 document.

20 DR. MARTY: Um-hmm.

21 DR. FUCALORO: It's a quarter of what's there.

22 CHAIRMAN FROINES: .014?

23 DR. FUCALORO: .014.

24 CHAIRMAN FROINES: Or .14?

25 DR. FUCALORO: .014. Unless it's an error.

1 That's always a possibility.

2 DR. MARTY: What page?

3 DR. FUCALORO: Look on page 2. Unless I'm

4 reading it wrong.

5 DR. MARTY: I know it's lower than the .05 but

6 I can't -- oh. Okay. According to CARB's monitoring

7 network, they are saying the ambient air lead

8 concentration in California in '99 -- that would be a

9 mean -- was .014.

10 I think the point is that there -- existing

11 blood lead levels in children, there is a concern adding

12 more lead into the air of pushing more kids above the

13 level of concern as identified by the CDC of 10

14 micrograms per deciliter. That's really the point of

15 this.

16 DR. FUCALORO: Yeah. I think that's the thing

17 that's a little confusing in the sense of reading it

18 that the ambient air concentration does not seem to

19 explain the level of lead in the blood. So one can

20 infer from that, one may infer from, that they're

21 getting lead into their system in other ways. Ingestion

22 or --

23 DR. MARTY: There's no argument that they're

24 getting lead from lead paint ingestion --

25 DR. FUCALORO: Yeah.

1 DR. MARTY: -- and other sources.

2 DR. FUCALORO: Right.

3 DR. MARTY: There's no argument there. If

4 DR. FUCALORO: Soil, picking it up.

5 DR. MARTY: Right. What we're concerned about

6 is twofold. Additional lead sources emitting into the

7 air. It's not -- as you now, as you can see from this

8 information, lead exposure is on a regional basis, which

9 is what the air monitoring network gets at are probably

10 not much of an issue. We are concerned with hot spots

11 of exposure.

12 DR. FUCALORO: An average is only an average.

13 I mean, the distribution of values is the most

14 important. I understand.

15 DR. WINDER: Okay. So next slide, please.

16 Looking at some of the more recent data to address the

17 question of whether or not negative effects associated

18 with blood lead levels below the 10 micrograms per

19 deciliter occur.

20 These are a couple of studies. In the top one,

21 it's a little bit complicated to explain here, but in

22 the top one, Campagne et al., we're looking at both the

23 mother and cord blood activities of calmodulin-

24 stimulated calcium pump activity. So what we're looking

25 at here is measuring lead levels in mother's hair, in

1 cord blood and in the newborn's hair.

2 What we see over here in the left-hand side,
3 they did this experiment looking at both the calcium
4 pump activity unstimulated with calmodulin and then the
5 bottom two rows were stimulated with calmodulin to see
6 if there's an effect of the stimulatory property and
7 broke it down into the level at less than .7, and this
8 is looking at the lead in the newborn's hair, .701, .5
9 and greater than 1.5.

10 And what we see here is that, according to this
11 series of studies, if you look at the cord level
12 unstimulated with calmodulin and as stimulated with
13 calmodulin, we find that there's a pretty significant
14 decrease in the calcium pump activity associated with
15 increases in the lead.

16 Now, the second graph in the bottom is trying
17 to give you a handle on -- since the top graph is
18 looking at lead in infant hair, in the mother's hair,
19 the bottom one is giving you a feel as to what that
20 corresponds to in blood lead. So, for example, in the
21 cord blood on the right-hand column in the bottom graph,
22 the cord blood was showing the lead at 4.8 micrograms
23 per deciliter, and that corresponded to 1.1 micrograms
24 per gram of lead in the newborn's hair.

25 And so you see this level of 1.1 is right in

1 that middle set up there in the top column. So what
2 this is showing is that the effects that we're seeing,
3 in this case the inhibition of the calcium pump, are
4 happening at blood levels well below the 10 micrograms
5 per deciliter level. Roughly half. So this is
6 suggestive evidence that the -- that 10 micrograms per
7 deciliter may be too high.

8 Now, there's an additional study which was
9 looking at the -- unfortunately, we don't have a slide
10 on this one. A gentleman was looking at the brain stem,
11 auditory and vocal response, which is commonly used in a
12 lot of these neurotoxicology studies.

13 And, again, he was finding that in children
14 with blood lead levels below the 10 micrograms per
15 deciliter, that is, from zero to seven and seven
16 micrograms per deciliter up, they were seeing effects
17 on -- let's say they evoked a response. That is to say,
18 increasing lead increased the conduction interval
19 associated with this DRE.

20 As the blood lead levels rose higher, the
21 conduction interval got shorter. We don't know why that
22 is. The authors are speaking that lead is, in fact, at
23 low levels inhibiting the growth of the neurites. And
24 at other levels in addition to that may be affecting the
25 myelination.

1 The upshot that the researcher gives is that
2 these levels -- these effects are being seen at less
3 than the 10 micrograms per deciliter.

4 DR. MARTY: I think part of our point is that
5 we are currently treating non-cancer health effects of
6 lead and no threshold phenomonon, at least at
7 concentrations that we can observe in our modern
8 environment. And there continues to be information that
9 you can see effects, at least at the biochemical level
10 and at the cellular level at concentrations below
11 10 micrograms per deciliter.

12 CHAIRMAN FROINES: Do you have any idea what
13 the concentration of lead in the air in Los Angeles is?
14 Because the .014 is a California-wide. It's clearly
15 going to be different in an urban environment.

16 DR. MARTY: We can look that up.

17 Jim, do you happen to know by any chance?

18 DR. FUCALORO: While they're looking it up,
19 very often my place has students study the soil.
20 There's a lot of lead in the soil. It's still there.
21 Not surprisingly I guess.

22 CHAIRMAN FROINES: Well, they still use leaded
23 oil, leaded fuel in airplanes.

24 DR. MARTY: We were looking at some of the
25 information from the Air Toxics Hot Spots Risk

1 Assessments that we received from the facilities
2 emitting lead into the air. In a couple cases, we did
3 get one hour maximum modeled concentrations that were
4 considerably above the existing standard ambient air
5 quality standard, which is a hard comparison to make
6 because that's a 30-day average in time.

7 One of them was about 5 micrograms per cubic
8 meter. There was an earlier risk assessment that we saw
9 way back in 1990 where they had model concentrations as
10 high as 50 micrograms per cubic meter for a one hour
11 max. So we are --

12 CHAIRMAN FROINES: But those are out of
13 secondary smelters I bet, aren't they?

14 DR. COLLINS: This was a battery company.

15 DR. MARTY: Battery.

16 CHAIRMAN FROINES: Secondary smelter, battery
17 company.

18 DR. MARTY: Right. Right. So we still have a
19 concern about hot spot exposures. And, in addition, we
20 have a concern about the no threshold phenomenon and
21 adding additional lead burden to the -- to kids
22 particularly.

23 CHAIRMAN FROINES: Well, I think that if you
24 look at the airborne concentrations of lead in the L.A.
25 Basin you would probably -- and then run it through the

1 various models, whichever ones you choose, you'll find a
2 fair percentage of kids predicted to have blood leads
3 over ten.

4 DR. MARTY: That also goes by race and
5 ethnicity. African-American kids have higher blood
6 levels from --

7 DR. FUCALORO: Environment.

8 DR. MARTY: Right. Right. So they, as a
9 population, are a sub population of kids who are
10 particularly at risk.

11 CHAIRMAN FROINES: Comments? Questions?
12 Mercury.

13 DR. GLANTZ: I guess I have one quick comment.
14 I think in terms of the placement as one of the five,
15 lead is pretty uncontroversial.

16 CHAIRMAN FROINES: It would have a high
17 ridicule value not to show up on the list.

18 DR. GLANTZ: Yeah.

19 DR. WINDER: Okay. So in talking about
20 mercury, mercury was put on Tier 2 as opposed to Tier 1.
21 The reason for considering it on List 2, again, it's a
22 neurotoxicant with a fairly well-defined series of
23 symptoms. Again these manifest themselves primarily in
24 young children.

25 A lot of the studies that you find published

1 deal with methylmercury exposure both in utero and
2 postnatally, and these effects are seen at levels that
3 are far below those especially for adults.

4 The reason for considering it Tier 2 as opposed
5 to Tier 1, is that in California at least, air is a
6 relatively minor transport medium for mercury.

7 Now, next supplied, please. The evidence for
8 this differential effect in children versus adults.
9 Much of this again derives from methylmercury data on
10 children. In this case, we're looking at Minamata, a
11 disease in Japan. Where children were displaying
12 this -- this I'll describe as congenital cerebral palsy,
13 and their lead -- I mean, their mercury concentration
14 hair was, as you see the range here, 5.22 to 110 parts
15 per million.

16 Now, in that same group, the mothers were
17 examined, and their maternal hair, as you see below
18 that, is over a somewhat broader range and generally a
19 little bit higher. The significant thing here is that
20 the children were expressing fairly severe symptoms.
21 These included mental retardation, ataxia, limb
22 deformities and may cases -- or in some cases death.

23 Whereas for the mothers, their symptoms were
24 usually paresthesia, fairly mild tremors, limb pains,
25 this kind of stuff. So -- and there are a number of

1 other reports, not just from Japan but elsewhere. For
2 example, the Iraqi studies, which suggest that, again,
3 very often mothers who present as having few or no
4 symptoms and yet have severely affected children.

5 Now, as with lead, again, the same kind of
6 concerns about children's behavior being one of the
7 things that figures into this higher exposure.

8 Now, the next slide, please. In this study,
9 this is by Marsh et al. This is looking at the mothers
10 and children -- mother and children pairs in Iraq that
11 were exposed to lead treated -- excuse me,
12 mercury-treated grain. And in this particular instance,
13 what we're looking at is the -- the kids were examined
14 in several different categories, looking at motor
15 effects, looking at the effects of mercury in speech,
16 mental performance and frequency of seizures.

17 Now, this particular graph is broken up into
18 the mercury levels seen in the mother's hair. Now, what
19 this shows, in all cases, the dark blue bar is
20 significantly higher than the rest, showing that at the
21 higher levels of mercury in the mom's hair, 99 to 384
22 parts per million, there is substantially greater
23 representation of the children with these motor defects,
24 deficits in speech, performance in mental tests and
25 frequencies of seizures. And the significance levels of

1 these things are at the .01, .001 levels.

2 DR. BLANC: I think you can probably go fairly
3 rapidly through the slides on the pediatric sensitivity
4 to mercury. I don't think there are going to be any --
5 so all of it's going to revolve around how you
6 approached the potential for airborne exposure and how
7 small, theoretically, an incremental exposure would have
8 to be for something for which you would imagine that the
9 bulk of the exposure is perhaps through diet, but
10 whether or not you think any increment would be relevant
11 or what -- how small an increment it would have to be to
12 be relevant.

13 DR. MARTY: Let's move to the exposure slides
14 then. Is that okay with you, Bruce? Or do you have
15 slides that are relevant to the question?

16 DR. WINDER: Well, these are again slides that
17 look at the effects associated with mercury.

18 DR. MARTY: Okay. I think we've established
19 that kids are more sensitive to it than adults. The
20 reason we ended up putting it on Tier 2 is because of
21 what we talked about earlier, that airborne exposures,
22 at least on a regional basis, don't appear to be
23 contributing a lot to total mercury intake.

24 We did come up with some information from --

25 DR. WINDER: This is from -- the presentation

1 is on the screen right now. This is some data from a
2 very recent meeting in San Francisco sponsored by EPA.
3 They're looking at mercury emissions from various mine
4 sites around the state. These typically are mines that
5 are no longer active. They were once involved in gold
6 mining, in some cases. Subsequently, mercury mines.

7 So what you see here is this sulphur bank mine,
8 for example. They show a flux of mercury of 922
9 nanograms per meter squared per hour. In that
10 particular mine situation, the authors calculate based
11 on the actual exposed surface area that there's an
12 annual flux of about 6.5 kilograms per year of mercury
13 into the air.

14 Down into the McLaughlin Gold Mine, this is
15 broken up into two areas, the pit, which is the actual
16 mining area is, as you see, putting out some 674
17 nanograms per meter squared per hour. Whereas the mine
18 tailings, which include mercury associated with
19 extraction of the gold, putting out somewhat higher than
20 1,000 nanograms per meter squared per hour.

21 So this gives a calculated flux for the -- both
22 areas around 15 kilograms per year, which comes out to
23 around 32 pounds per year. Now, that's substantially
24 higher than what the ARB tells us lead emissions by
25 facility are. Those -- particularly for the state.

1 Excuse me mercury emissions by the facility,
2 particularly for the state, are limited to around
3 6 pounds.

4 So there are some, as Melanie put it, some hot
5 spots of mercury vapor throughout California.

6 DR. MARTY: It would be nice if we had a nice
7 model like the IEUBK model, which relates blood air
8 concentrations to -- blood lead concentrations to air
9 lead concentrations. We don't have a similar model for
10 mercury.

11 Nonetheless, the concentrations measured in air
12 are around -- in the nanogram per cubic meter amounts
13 regionally. Bruce has an example where it would
14 certainly be higher than that judging by the emissions
15 rates that you see on the screen.

16 So, again, it's not a regional problem, may be
17 a hot spots problem, but the concentrations still are
18 relatively low.

19 DR. BLANC: Do you have a -- a main priority
20 cutoff for how many hot spots there need to be for
21 something to raise up in your prioritization based on
22 hot spots once you know that the ambient levels are not
23 the issue?

24 DR. MARTY: We don't.

25 DR. BLANC: Is -- would ten be too many? Are

1 five too few?

2 DR. MARTY: We actually didn't discuss that.

3 DR. BLANC: And do you have --

4 DR. MARTY: In terms of lead, there's probably

5 around five.

6 DR. BLANC: So that was enough for you for

7 that?

8 DR. MARTY: For mercury, you do get mercury

9 emissions from, for example, municipal and hospital

10 waste combustion processes since it's not trapped.

11 DR. BLANC: That's what I wanted to ask about

12 specifically. So do you have a level monitoring data

13 that tell you what the emissions are near hot spots that

14 have medical waste incineration?

15 DR. MARTY: We don't have monitoring data.

16 There are some modeling studies that have been done

17 looking at mercury from medical waste incinerators

18 primarily. We can look at some of that.

19 DR. BLANC: Does your document list how many

20 medical waste -- licensed medical waste incinerators

21 there are in the State of California?

22 DR. MARTY: No. No.

23 DR. BLANC: Wouldn't that be something you

24 would want?

25 DR. MARTY: Yes. There's far fewer than there

1 used to be because the dioxin airborne toxic control
2 measure really forced people to stop burning medical
3 waste onsite and instead transport it to a state-of-the-
4 art regional facility.

5 DR. BLANC: But mercury is not captured in
6 those; right?

7 DR. MARTY: No, it's not.

8 DR. BLANC: So basically what you've done is
9 tightened the concentration at the hot spots but limited
10 the number of hot spots.

11 And do you have any ambient airborne monitoring
12 data from Santa Clara County in the areas near the
13 former Almaden mining operations?

14 DR. WINDER: No, I don't.

15 DR. BLANC: And have you contacted Santa Clara
16 County health officer to see if they have some control
17 over data that you might not be able earn?

18 DR. MARTY: No, we haven't done that. We
19 should do that.

20 DR. BLANC: That's the largest mercury mine in
21 the world formerly. I think it would be worth it.

22 DR. MARTY: I think it definitely would be
23 worth looking at.

24 DR. BLANC: Again, it's kind of the parallel to
25 your argument on lead. But since you're going to put in

1 the top five, I think you're not going to get any
2 argument from us here. And, clearly, mercury is, in
3 your group, getting very close consideration, and you're
4 not going to get any argument about that either.

5 The question is, Have you met enough of a
6 burden of disproof? Have you proved the negative enough
7 to satisfy yourself that it shouldn't be among the five
8 or at least it's outweighed by the things that you have
9 chosen? And, you know, I think that's going to be
10 something that we're going to have to look at closely.

11 DR. MARTY: We'll have to be -- to bolster that
12 explanation --

13 DR. BLANC: Yes.

14 DR. MARTY: -- in this document.

15 DR. BLANC: Because I'd hate to have us miss
16 the boat on that just because we didn't ask the right
17 questions.

18 DR. MARTY: Right.

19 DR. BLANC: And, you know, again, it's all
20 relative, but if we're looking at the -- and we're not
21 going to go into the other four things. What table is
22 the final one? I'm sorry.

23 DR. MARTY: It's Table 1, page 8.

24 DR. BLANC: What page is it on?

25 DR. GLANTZ: We may have time to get one or two

1 more.

2 DR. MARTY: Page 8.

3 DR. FUCALORO: Dioxins.

4 DR. BLANC: Yeah. But let's say we're looking
5 at dioxins and PCBs; right? Now, you've said -- you've
6 just said that, for example, for medical waste, the
7 dioxins at least are being destroyed by the temperature
8 pheresis.

9 DR. MARTY: Not entirely. But yes, the idea
10 was to reduce the emissions.

11 DR. BLANC: Whereas we know that mercury is not
12 being touched.

13 DR. MARTY: Yes

14 DR. BLANC: And is not being captured. So in
15 terms of this, you know -- and with dioxins, we're
16 really not talking about ambient concentrations either,
17 I don't suppose. We're talking about hot spots also;
18 aren't we?

19 DR. MARTY: It's both. It's regional exposures
20 and hot spots for dioxin.

21 DR. BLANC: Well, maybe those are the two. We
22 were sort of inherently pairing lead and mercury in this
23 discussion, but maybe the discussion is more parallel
24 for dioxins and for mercury.

25 CHAIRMAN FROINES: Do you know, by the way, if

1 there are any mercury thermometer plants in California?

2 DR. MARTY: I don't know. I don't know. That
3 we can ask ARB. We can try to figure out how many
4 facilities there are emitting mercury also in the hot
5 spots database. You just add them up and where they
6 are.

7 I think it's fair to point out, though, that
8 the, quote, "mercury problem in California" is because
9 we mined it in the foothills, we dredged it -- trucked
10 it across the valley and used it for gold mining in the
11 Sierras, so we contaminated a lot of streams, and it's
12 since run down and just spread itself all over the
13 foothills and the valley, contaminating food sources for
14 people. So that's a pretty important exposure for
15 mercury.

16 DR. BLANC: I know it's an incremental issue
17 you're dealing with.

18 DR. MARTY: Yes.

19 DR. BLANC: But your statute clearly tells you
20 that you need to take that into account, and it doesn't
21 really matter whether the air source is the bail of hay
22 or it's the straw that's breaking the camel's back.
23 Either way, you need to deal with that, and that makes
24 your life pretty complicated.

25 But still, for this one, it's proving the

1 negative argument I think that's going to be critical
2 and not simply saying -- since you already have
3 disproved the validity of the -- in the release
4 inventory; right? Your slide on the mines, those mines
5 are in California?

6 DR. WINDER: Yes.

7 DR. BLANC: So you already know that there's
8 far more mercury going up than the release inventory
9 tells you is going up; right?

10 DR. WINDER: Yes. I mean, our inventory for
11 the state was something like 6,400 pounds or thereabouts
12 per year. And as you see from this for example,
13 McLaughlin, it was about 33 pounds per year. So it's a
14 small portion of that, but your point is well taken with
15 regard to the slides.

16 DR. FUCALORO: It would helpful -- I asked Paul
17 this. Would it be helpful to play mercury off against
18 lead? They both seem to have the same sorts of things.
19 They're both extremely toxic, and their exposure level
20 is low now and probably getting lower. And one of them
21 is going to make the first tier and the other is going
22 to make the second tier. So would a comparison between
23 those two be useful?

24 Paul, I asked you that question.

25 DR. BLANC: What I was saying was maybe not.

1 Maybe the comparison should be between dioxin and
2 mercury.

3 DR. FUCALORO: You're thinking dioxin. Sorry.

4 DR. BLANC: Well, you can say that it's the
5 obvious one, but maybe it's not so obvious.

6 DR. MARTY: Yeah. And the natural inclination
7 is to look at the two metals that are developmental
8 neurotoxins in humans. Well documented.

9 DR. BLANC: But the real issue is that you have
10 two substances in the group -- in the top group, both of
11 which everybody is going to say is not of the big
12 player. They both made it into the top 11 one way or
13 the other. That's dioxin and mercury. But they're the
14 one for which the air exposure data are the lowest of
15 all these but they both --

16 DR. MARTY: I think maybe a little bit of -- in
17 the case of dioxin, almost all the dioxin that ends up
18 in the food chain initially was airborne from combustion
19 sources. Bleaching of pulp during paper making used to
20 be a significant source and is responsible for a lot of
21 the residual that you see near pulp mills. But,
22 currently, the dioxin that enters the food chain came
23 out of some combustion process somewhere.

24 You can't really -- so we viewed it as, okay,
25 the problem is controlling it from coming out in the

1 first place. In the case of mercury, it's really --
2 it's a little bit different in that the primary sources
3 are water born, not initially airborne. So that's one
4 thing that we weighed when we looked at aggregate
5 exposures.

6 DR. BLANC: Except that you have no way of
7 controlling the dioxins probably.

8 DR. MARTY: It's sure getting a lot of
9 attention at U.S. EPA and also at CPAAPCO, the
10 California Association of Air Pollution Control Officers
11 have a project they're doing, trying to figure out if
12 residential burning in California, and that's, you know,
13 burn barrels is a significant source of dioxin. So they
14 are trying to focus a little more on where the dioxin is
15 coming from.

16 There's a lot of papers on global flux of
17 dioxin, and it seems that there's more that you can
18 measure out there than you can account for in terms of
19 emissions. So it's -- which is -- it's a tricky thing
20 to do but --

21 DR. FUCALORO: Naturally occurring.

22 DR. MARTY: Lots of people are looking for
23 where is it all coming from? And also I should add that
24 ARB did look at our list and didn't flinch at -- when
25 they saw that dioxins was in the top tier. You know,

1 some of the comment we got from them indicated that they
2 thought they could do more to control dioxin.

3 CHAIRMAN FROINES: I think that that's a --
4 Paul's also raising a generic issue within the context
5 of the specific one, which is when we get down to the
6 final five, I think we'll need -- we want to have a
7 clear discussion as to how the ultimate selections were
8 made relative to each other.

9 And this points out -- the issue of dioxins
10 versus lead versus mercury points out that you have on
11 the one hand the strength of the evidence, and the
12 second is, of course, the exposure, and those two will
13 probably be the defining features. But, in general,
14 we'll have to make sure that those are well described.

15 My guess is that this is a good time to stop
16 for the day. I don't think we should take up another
17 chemical.

18 DR. FUCALORO: Good guess.

19 DR. BLANC: Yeah. Good.

20 CHAIRMAN FROINES: Can we have a motion to
21 adjourn?

22 DR. GLANTZ: So moved.

23 DR. BLANC: Second.

24 CHAIRMAN FROINES: All in favor?

25 ALL: Aye.

1 CHAIRMAN FROINES: The meeting is officially
2 closed for April 27th, 2001, with the Chair's thanks to
3 everybody who participated.

4 (Proceedings concluded at 4:40 p.m.)

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4 I, Jennifer S. Barron, CSR 10992, a Certified
5 Shorthand Reporter in and for the State of California,
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14 I further certify that I have no interest in
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